American Journal of Diseases of Children

VOLUME 32 DECEMBER, 1926 NUMBER 6

THE TREATMENT OF GONORRHEAL VULVO-VAGINITIS IN INFANTS BY MEANS OF VACCINES *

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REVIEW OF THE LITERATURE

The use of vaccine therapy in gonorrheal genital infections dates back a considerable period. It was Wright who, as a result of his investigation, first utilized the gonococcus vaccine. With reference to this vaccine, Jarvis [1] makes this statement: "Wright's vaccines are microbes, killed by heating at 60 C. for half an hour. The vaccine material consists of intracellular toxins of the bacteria, as the protoplasm has been inactivated by heat."

It is apparent that the original idea of Wright was to provoke an antitoxic action, based on the supposition that the gonococcus particularly acted through the secretion it produced.

At a later period, Mainini used other vaccines which were heated to 70 C. and then titered by means of a Thomas-Zeiss hematometer. Mauter [2] employed aqueous autogenous vaccines heated only to 53 C. He felt that higher temperatures diminish the power of the vaccine. As a dosage he used from 5 to 15 million germs per cubic centimeter. Jarvis, who has done a great deal of work in this field, and who has carefully analyzed the works of Irons, Whitmore, Eyre and Stewart, comes to the conclusion that the results with vaccine treatment of gonorrhea are variable and quite uncertain. Sometimes they are excellent and again they are absolutely nil. Other investigators, such as Bristow, Cole, Meakins, Brucke, MacOscar, Dieulafoy, Maute, Renaud and Bedet, have come to similar conclusions.

At a later date, Besredka and Cruveilhier reported favorable results in a considerable number of cases of gonorrheal orchitis, cystitis and vulvovaginitis. Recently, a vaccine prepared by Nicoll and Blaizot

* Received for publication, Aug. 5, 1926.

1. Jarvis: Presse méd., 1910.
2. Mauter: Soc. de Biol. 27:3, 1909; Presse méd., 1911.

aroused quite a bit of interest. These authors reasoned that the toxic products of the gonococcus were the principal obstacles in the way of its safe employment, and that the antiseptics and heat employed in the preparation of the vaccine caused too much alteration in the bacterial protoplasm. They attempted to avoid both difficulties, and with this end in view they sterilized the gonococcus emulsion by using sodium fluoride. This treatment produced few alterations in the protoplasm. The emulsion is then centrifugalized and washed with physiologic sodium chloride solution. In this manner it is freed entirely from all its toxic products. Nicoll, also, for the first time, used a small gram-positive diplococcus along with the gonococcus. He gives it the name, "synococcus." He advanced the theory that this diplococcus, although nontoxic by itself, is a factor, if associated with the gonococcus, in causing gonorrheal infection to persist. There is much division of opinion regarding the effectiveness of the vaccine of Nicoll and Blaizot. Some observers, notably Nicolas, Favre, Gaté, Legueu, Nogues, Moras, Lebreton, Minet and Debains, are adverse in their criticism, while others, particularly Moreau, Dubois, Bonamour and Terrisse, report a number of cures with its use.

The studies of Professor Widal on colloidal shock appeared about this time. They explained many therapeutic phenomena which heretofore had escaped observation. According to Widal and his followers, the action of the vaccine has considerable resemblance to this shock, and its efficacy may be due to such protein shock. The nature of the antigen is unimportant. The favorable effect is produced by any one protein present. Nonspecific proteins, such as ovo-albumin, sero-albumin, gelatin, histamine, Witte peptone or milk, were recommended and used for this purpose. The promiscuous application of this theory of Widal led to much confusion and wrong deductions. The theory of Widal is doubtless one of the most valuable contributions to immunotherapy, but accepting the specific character of certain organic reactions (proof is furnished by the action of antityphoid vaccine, which does not immunize against paratyphoid A infection, and this, in turn, does not immunize against paratyphoid B) it is, nevertheless, necessary to qualify this acceptance with considerable reserve.

Lebeuf explains the cure of chronic conditions by means of vaccines prepared with nonspecific proteins on the basis of two kinds of organic reactions. One is a general or group reaction which is produced by any protein; the other is a special reaction which responds only to the specific protein. Logically, bacteriologists should take advantage of both reactions. Together and in properly balanced association they should react more effectively than if operating alone and independently.

During recent times American writers, particularly Butler and Long [3] Hamilton,[4] Cook and Churchill and Soper,[5] have devoted much study to affections of the urogenital tract of the female child and to the use of vaccine therapy in vulvovaginitis of infancy and childhood.

We have been interested for the past two years in the vaccine therapy of a number of infectious diseases. Previous to this time (since 1910), one of us (Morales) had been experimenting with autogenous vaccines and studying their effect. Among other conditions we studied its effect on vulvovaginitis of children, quite unaware at the time, and therefore independent of similar research carried on by Dr. Luisa Bonacorci. She records the interesting observation that a distinct difference exists between the gonococcus found in female children and that found in adults. These differeces are biologic in nature, and consist of difference in agglutination reaction and complement fixation. Both are more pronounced and have a greater titer with homologous serums. In much the same way an antigen prepared with the gonococcus of the adult fixes complement only with the serum of adults and not with the serum of a female child. Conversely, the antigen prepared with the gonococcus from a female child fixes complement only with serum from the child and not with serum from an adult.

Our own results are in accord with those of Dr. Bonacorci, and are substantiated by clinical proof and experience as set forth in the appended case histories. Thompson and Vollmond and Luise Pearce and Maja have all been able to find and confirm this striking biologic difference in agglutination reactions and complement fixation between the gonococcus of vulvovaginitis of the female child and the gonococcus of the adult.

The existence within the type "gono" of strains with particular attributes is not a new discovery. Dopter and Sacquepe, Kolle and Hoetch Parisot and Simonin mentioned this fact. Bruckner and Cristenu disagree with their observations. Torrey believes that there are a number of different gonococci. Watabiki, working with eight types, found that he could divide them into two groups. Jotten, who studied twenty-five specimens with reference to agglutination and complement fixation, found four groups with clearly defined characteristics. Hermanies, in a study of eighty-five specimens, found six distinct serologic groups. All these different researches go to show that the conception of various strains of gonococci is not a new one. Viewed in the light of these experiences, the observations by Dr. Bonacorci of

3. Butler, W. J., and Long, J. P.: The Vaccine Treatment of Vulvovaginitis in Children, J. A. M. A. **50**:744 (March 7) 1908.

4. Hamilton, B. W.: Gonococcus Vulvovaginitis in Children, J. A. M. A. **54**:1196 (April 9) 1910.

5. Churchill, F. S., and Soper, A. C.: The Inoculation Treatment of Gonococcus Vulvovaginitis in Children, J. A. M. A. **51**:1298 (Oct. 17) 1908.

differences between the gonococcus from the female child and that from the adult are conceivably correct, and are probably largely due to modifications that the germ is subject to, owing to the difference of mediums in which it develops. To us, the fact seems important that autogenous vaccines have undeniable curative properties. In our experience it is possible with their use to subdue in a relatively short time a disease which up to the present has tenaciously resisted all forms of therapy.

TECHNIC OF PREPARING THE VACCINE

We have utilized not only the cultures of gonococci isolated in each case but also a portion of the accompanying flora. It is well known that gonorrheal disease of the female in its early period differs from that of the male, in that the organism is not present in pure state but is always associated with other bacteria such as colon, streptococcus, staphylococcus and, very occasionally, proteus. The culture is obtained in the following manner: The vulva is washed with physiologic sodium chloride solution forty-eight hours before taking the specimen. A platinum loop full of thick purulent discharge is then taken from some point near the urinary meatus and inoculated on blood agar plates or on albumin agar. The plates are placed in the incubator for twenty-four hours.

Isolation of the gonococcus in the pure state is not easy, on account of admixture with other germs which grow more rapidly and easily. In order to accomplish it, we proceed as follows: The plate containing the least colonies is selected. Of these colonies the smallest, most transparent and least developed is selected. This is transplanted to a tube containing egg-protein agar. At intervals of 1 cc., other similar colonies are transferred until four or five colonies have been inoculated in each tube. Four tubes are generally sufficient for twenty colonies. Among this number one or more will show the gonococcus in pure state. The tubes are left in the incubator and on the following day the colonies are identified. Only such as are gram-negative are selected. The colonies which are most abundant in the plates are transplanted to agar tubes identified on the following day and titration is begun. To titer the bacteria an apparatus known as a nephelometer is employed. Its accuracy is sufficient for practical purposes. The vaccines are titered in three series, containing respectively 125, 250 and 500 million germs per cubic centimeter. A vaccine, for example, may have the following composition in case the plates contained colon bacilli, staphylococci and streptococci besides the gonococcus:

Gonococci, 80 million; colon bacilli, 10 million; staphylococci, 15 million; streptococci, 10 million, or a total of 115 million. Numbers two and three show a similar relationship.

If the bacteria vary from the ones mentioned, we proceed more or less in much the same way. After titration of the vaccine, it is sterilized

at 55 C. for one-half hour, then placed in the mechanical shaker and emulsified. It is then put into a series of 6 ampules, each containing 1 cc.

Treatment is begun by injecting subcutaneously the contents of one ampule of series 1. Within from twenty-four to forty-eight hours, depending on the reaction, the contents of all or part of the rest of series 1 is injected. This is followed at a proper interval by injection of vaccine of series 2 and 3. The dosage may be increased if necessary. In some cases it is well to have a rest period of eight days between the serial injections.

Our results have been very good, not only with the autogenous vaccine but also with stock vaccine, provided the source of the material was a female child. The secretion generally diminished considerably a few days after beginning the treatment. It often ceases entirely before completion of the third series of injections. Bacteriologic control shows the disappearance of the gonococcus and also appreciable diminution of the colonies of other organisms. The following cases will illustrate the striking results of the treatment:

REPORT OF CASES

CASE 1.—Z. O., a girl, aged 8 years, had a discharge which had been noticed only three days prior to examination. The mother had some discharge. Bacteriologic examination and culture revealed the presence of gonococci in the discharge of the child. An autogenous vaccine was prepared. Injections of this vaccine were given over a period of five weeks, beginning with 125 million germs for the first injection and ending with a dosage of 1,500 million. At the end of the fourth week the gonococcus had disappeared from the secretion.

CASE 2.—N. F., a girl, aged 7 years, had a discharge which was noted eleven days prior to examination. The mother had a greenish yellow vaginal discharge. Bacteriologic examination of the discharge from the child showed the presence of gonococci. An autogenous vaccine was prepared from the culture. The treatment was continued over a period of eight weeks. The vaccine was given in dosage of from 125 million to 1,500 million germs. At the end of the fifth week the gonococcus had disappeared from the secretion. The child had remained cured up to the time of writing.

CASE 3.—Z. S., a girl, aged 11 years, had had a discharge for one year. The mother had had a discharge for the past eight years. Microscopic examination and culture showed the presence of the gonococcus. An autogenous vaccine was prepared and given in dosage of from 125 million to 3,000 million germs over a period of eight weeks. No painful reactions were observed. Complete cure was effected within eight weeks. The child had remained well up to the time of writing.

CASE 4.—In R. C., a girl, aged 6 years, a sister of Z. S. (case 3), the discharge was noted a few days prior to examination, and was evidently a contamination from the sister, as both children slept in the same bed. Gonococci were found in the discharge. A stock vaccine made from gonococci taken from a female child was used. Injection was given in dosage of from 125 million to 1,500 million germs over a period of four weeks. At the end of this period the discharge had ceased. It reappeared again after a few days, but was free from gonococci. In another week it ceased permanently and the child had remained well to the date of writing.

CASE 5.—A. A. C., a girl, aged 8 years, had had a greenish yellow discharge for two months. Micturition was not painful. At another clinic the child was given stock vaccine treatment with vaccine prepared from a gonococcus taken from an adult. The treatment was without result. Bacteriologic examination and culture showed gonococci present, with large numbers of other bacteria. Treatment was begun with a stock vaccine prepared from the gonococci of a female child. The injections ranged in dosage from 125 million to 2,000 million germs. In less than four weeks of treatemnt the gonococcus had entirely disappeared from the discharge which had practically ceased. It reappeared for a short period a week later, then ceased entirely up to the time of writing.

CASE 6.—C. T., a girl, aged 10 years, had had a discharge for one year. The mother had had a discharge since the time of her marriage. Local treatment had no results. The discharge was very abundant, of a greenish-yellow color, and culture showed gonococci. From these, an autogenous vaccine was prepared. Treatment continued over a period of four months, the dosage of the vaccine injected ranging between 125 million to 3,000 million germs. There were no painful reactions. After the third month no discharge was noticed, and bacteriologic examination showed complete absence of gonococci. A few weeks later the discharge reappeared and again showed gonococci. A new autogenous vaccine was prepared and the treatment was repeated, this time with permanent cure. Repeated bacteriologic examinations showed no gonococci.

CASE 7.—C. E. R., a girl, aged 3 years, had a discharge which began twenty days prior to examination, almost simultaneously with the beginning of a discharge in the mother. Bacteriologic examination showed gonococci. Injections of stock vaccine prepared from the gonococcus from a female child were given over a period of three and one-half months, in dosage of 125 million to 1,500 million germs. At the end of the third month all discharge had disappeared, gonococci were absent and the child had remained well up to the time of writing.

CASE 8.—C. M., a girl, aged 7 years, three weeks before examination developed a discharge. The mother had had a discharge for the past three years. Bacteriologic examination and culture showed gonococci present. An autogenous vaccine was prepared, and treatment was continued over four months. Injections were given in dosage of from 125 million to 3,000 million germs. After the fourth month all discharge ceased. No gonococci were present, and the child had remained well to the time of writing.

CASE 9.—D., a girl, aged 4 years, had had a discharge for the past six months. Bacteriologic examination showed gonococci present. Treatment continued for a period of two and one-half months. Stock vaccine prepared from gonococci from a female child was used. The quantity of the discharge had lessened considerably but was not free from gonococci. The patient left the clinic and treatment was not continued.

CASE 10.—L. F., a girl, aged 5 years, had a discharge for a month prior to examination. Bacteriologic examination showed the presence of gonococci. Treatment was carried out over a period of four months, with several series of stock vaccines prepared from gonococci from a female child. Injections were given in dosage of 125 million to 2,000 million germs. No local or general reactions were observed. Local treatment with permanganate of potash solution was continued at the same time. All discharge ceased after the third month of treatment and gonococci were no longer found.

CASE 11.—M. A. Z., a girl, aged 9 years, had a discharge for six months. Bacteriologic examination showed gonococci present. Treatment was begun with stock vaccine, prepared from a gonococcus from a female child. The dosage of the injections ranged from 125 million to 2,000 million germs. The patient was discharged entirely cured after the third month of treatment.

SUMMARY

Five patients were treated with autogenous vaccine; six were treated with stock vaccine. Of this total of eleven, ten patients were entirely cured. In six of the patients the cure was effected in from one to two weeks; in three, from two to four weeks, and in one patient it required six weeks before a complete cure was obtained.

GROWTH IN CHILDREN WITH DIABETES MELLITUS *

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INTRODUCTION

The metabolic problem in a child suffering from diabetes mellitus is far more difficult than that in the adult, since in the child one must provide not only for maintenance but also for growth. It is a nice problem to accomplish this and at the same time to preserve the carbohydrate burning function of the body.

With the advent of insulin, the prospects for the future of the child with diabetes brightened. It seemed to mean that one could give sufficient food to sustain normal life and to keep the patient sugar-free. Formerly, it had been necessary to rely on reduced diet or on starvation. Yet when it came to prescribing a definite number of grams of carbohydrate protein and fat in an individual case, and adjusting the dose of insulin to the needs of that patient, one was faced with questions not so easy to answer.

It was desired to promote growth by giving a diet as nearly normal as possible. Yet in doing this it was found that the child fattened to a degree which seemed dangerous. Hence the question arose with each case as to whether the child was too fat or too thin, and whether it was of average size for its age. Was it advisable to give a diet theoretically adequate from a pediatrician's point of view? What were the essential factors that would bring about the attainment of normal growth and function. What should be the relation of total calories, grams of carbohydrate, grams of protein and grams of fat to the stature of the child or to his age?

In order to answer these questions, it was first necessary to find criteria of growth and food supply. Although we have not succeeded to our complete satisfaction, we have chosen certain well-known standards of growth and food intake, and have studied the histories of our diabetic children with reference to these standards. Notwithstanding the fact that these standards may be in need of revision, our comparisons have been so suggestive that we are presenting certain of our observations.

This paper is based on a study of thirty-four cases of diabetes mellitus and one case of possible renal diabetes in children. Twenty-three of

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* Received for publication, May 4, 1926.

* From the Department of Medicine of the College of Physicians and Surgeons of Columbia University, and the Presbyterian Hospital.

* A portion of the case material used has been supplied through the kindness of Dr. H. Rawle Geyelin.

the patients are males; twelve are females. These cases as a group cover the entire period of childhood from infancy through puberty (table 1).

THEORETICAL CONSIDERATION OF GROWTH CURVE IN NORMAL CHILDREN

This subject, if fully considered, would require much more space than this paper warrants. Curves of growth or stature correlated chronologically may be found on the walls of almost any children's

TABLE 1.—*Period of Disease by Age*

Case	Period of Disease, Years	Years of Age 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
27	1 to 4	—	—	—	—																
2	2 to 5	..	—	—	—	—															
13	2 to 5	..	—	—	—	—															
8	3 to 9	..	..	—	—	—	—	—	—	—											
17	3 to 5	..	..	—	—	—															
1	4 to 10	..	..	..	—	—	—	—	—	—	—										
23	4 to 6	..	..	..	—	—															
24	4 to 7	..	..	..	—	—	—														
9	5 to 6	..	..	..	..	—															
11	5 to 15	..	..	..	..	—	—	—	—	—	—	—	—	—	—	—					
34	5 to 10	..	..	..	..	—	—	—	—	—	—										
3	6 to 12	..	..	..	..	..	—	—	—	—	—	—	—								
6	6 to 8	..	..	..	..	..	—	—	—												
14	7 to 9	..	..	..	..	..	..	—	—	—											
20	7 to 15	..	..	..	..	..	..	—	—	—	—	—	—	—	—	—					
25	7 to 10	..	..	..	..	..	..	—	—	—	—										
35	7 to 9	..	..	..	..	..	..	—	—	—											
10	8 to 11	..	..	..	..	..	..	..	—	—	—	—									
29	8 to 18	..	..	..	..	..	..	..	—	—	—	—	—	—	—	—	—	—	—		
30	8 to 11	..	..	..	..	..	..	..	—	—	—										
12	10 to 12	..	..	..	..	..	..	..	..	..	—	—									
31	10 to 15	..	..	..	..	..	..	..	..	..	—	—	—	—	—						
33	10 to 13	..	..	..	..	..	..	..	..	..	—	—	—								
19	11 to 14	..	..	..	..	..	..	..	..	..	..	—	—	—							
28	11 to 13	..	..	..	..	..	..	..	..	..	..	—	—	—							
32	11 to 14	..	..	..	..	..	..	..	..	..	..	—	—	—							
16	12 to 18	..	..	..	..	..	..	..	..	..	..	..	—	—	—	—	—	—	—		
22	12 to 19	..	..	..	..	..	..	..	..	..	..	..	—	—	—	—	—	—	—	—	
7	14 to 17	..	..	..	..	..	..	..	..	..	..	..	..	..	—	—	—				
15	14 to 16	..	..	..	..	..	..	..	..	..	..	..	..	..	—	—	—				
4	15 to 16	..	..	..	..	..	..	..	..	..	..	..	..	..	..	—	—				
18	15 to 20	..	..	..	..	..	..	..	..	..	..	..	..	..	..	—	—	—	—	—	—
5	16 to 20	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	—	—	—	—	—
21	16 to 19	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	—	—	—		
26	16 to 19	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	—	—	—		

clinic. We have chosen as our bases for comparison the curves of Stratz [1] and of Bardeen [2] (charts 1 and 2). Other data of interest, together with valuable bibliographies, are given by Baldwin,[3] Scheidt [4] and Davenport.[5]

1. Stratz, C. H.: Gestalt und Wachstum des Kindes, in Kruse und Selter. Die Gesundheitspflege des Kindes, Stuttgart Euke, 1914, pp. 7-28.

2. Bardeen, C. R.: The Height-Weight Index of Build in Relation to Volumetric Proportions and Surface Area of the Body During Postnatal Development, Carnegie Inst. Wash., Pub. 272.

3. Baldwin, Bird T.: Physical Growth and School Progress, U. S. Bureau of Education, Bull. 10; The Physical Growth of Children from Birth to Maturity, University of Iowa Studies in Child Welfare, 1, no. 1.

4. Scheidt, Walter: Allgemeine Rassenkunde, Lehmann, 1925.

5. Davenport, C. H.: Body Build and Its Inheritance, Carnegie Inst. Wash., Pub. 329.

Since the measurements on our children are restricted to height and weight alone, we have chosen to represent changes in growth graphically by two types of charts: (1) by plotting the height-weight index curve of Bardeen; (2) by plotting percentages of deviations from normal height and weight for a given age, according to the curves of Stratz.

1. *The Height-Weight Index Curve of Bardeen.*—This height-weight index is but roughly correlated to age. To quote Bardeen:

> Age is of interest chiefly as a measure of the stage in the life-cycle reached by the individual at the time the study of his structure is made. As such it is imperfect, since some individuals of the same essential life-cycle may vary greatly at different periods. Thus the studies of Roberts [6] (1878) show that the

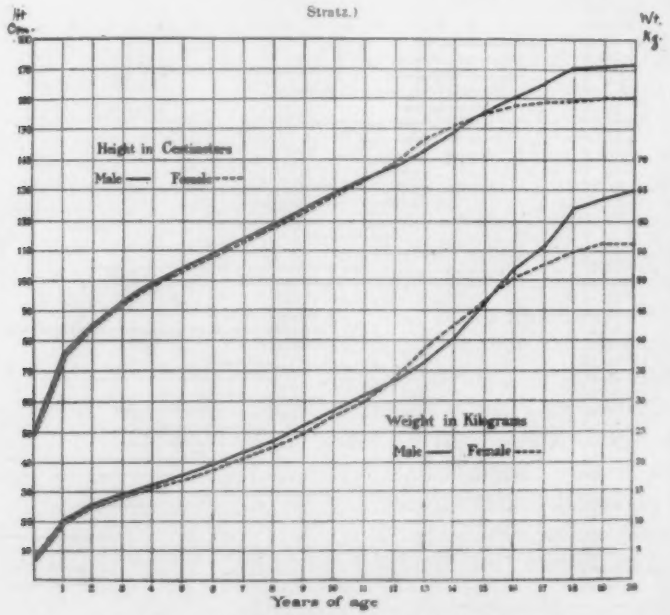

Chart 1.—Taken from Morris' Anatomy, 1923.

> children of the artisan classes in England grow more slowly than those of the wealthier classes up to adolescence, but that during adolescence, the former continue to grow for a longer period; so that, although the difference is not completely made up, there is at a given age less relative difference in size between adults of the two classes than between children of the two classes. The school children studied by Baldwin [8] grew on an average much faster than those studied by other observers up to adolescence, but the adolescents were of normal size.

Crampton [7] has made observations which support the opinion that there is a closer relation between the so-called physiologic ages, i. e.,

6. Roberts, C.: Manual of Anthropometry, 1878.

7. Crampton, C. W.: Physiological Age—a Fundamental Principle, Am. Phys. Educat. Rev., 1908, vol. 13.

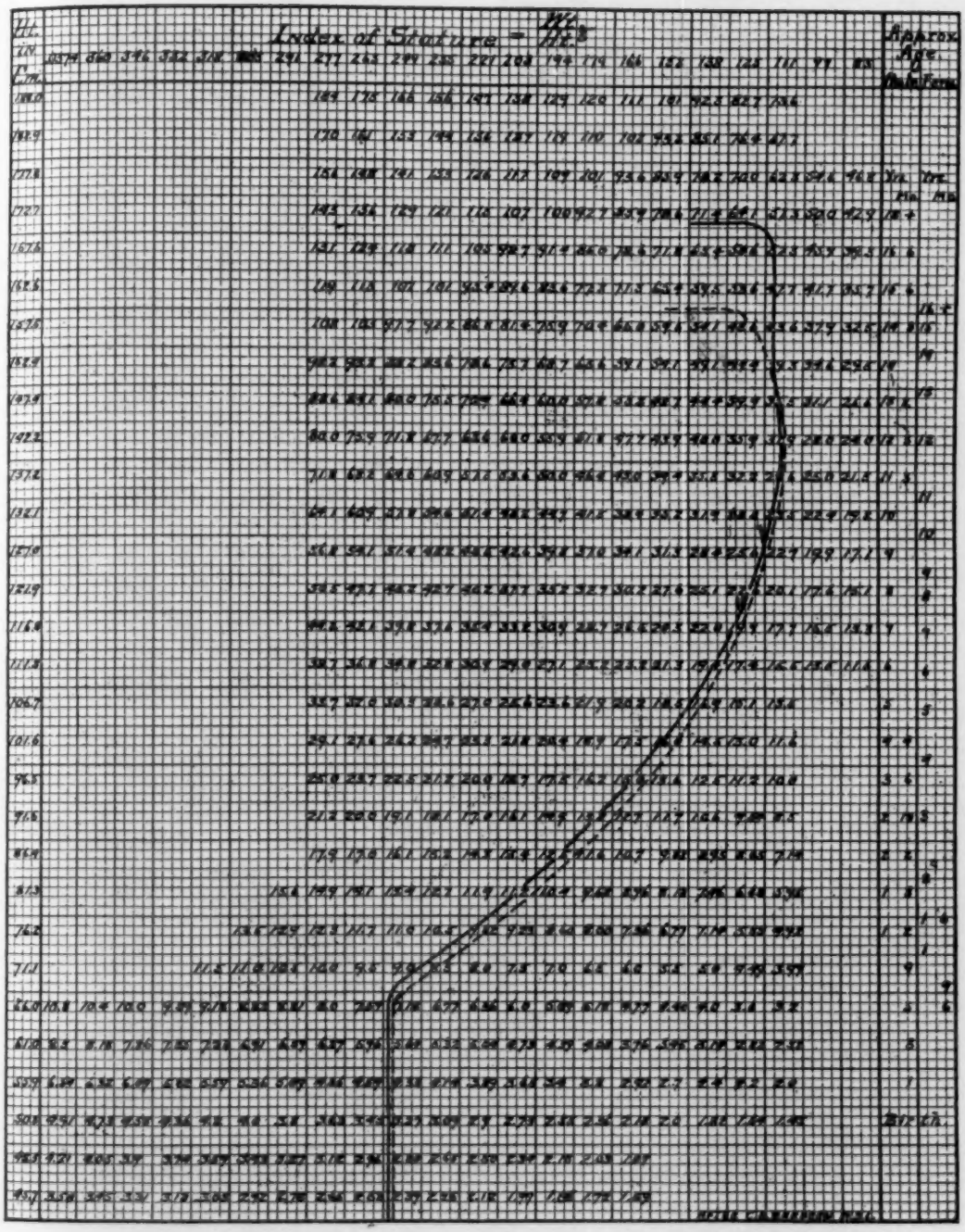

Chart 2.—Bardeen chart.

dentition, ossification, pubescence and growth, than between growth and the chronologic age. However, we have not the data of physiologic age, and for our purpose the rough correlation of chronologic age with the height-weight index must suffice for the time being.

Briefly, the basis of Bardeen's index is as follows:

> The volumes of objects of the same shape but of different sizes vary as the cube of a given diameter through these objects. The volumes of bodies of individuals of the same external form but of varying heights vary as the cube of the stature multiplied by a factor which is conditioned by the form of the body as π is conditioned by the form of the sphere. This factor in the case of the relation of volume to stature expresses the part of the space equal to the cube of the height occupied by the volume of the body.

Therefore, two persons of the same form but of varying heights would have the same index; conversely, if the ratio of the volume of the body to the cube of the heights varies, the body forms of the two persons must differ. Hence, if one occupies a greater part of the space equal to the cube of his height than another, the first must have a relatively greater transverse section.

The average specific gravity of the body is 1.032, which is the same as water at body temperature. One pound of body substance, therefore, occupies about 27 cubic inches. Hence, a man of 150 pounds (68 Kg.) weight would have a volume of 4,050 cubic inches. If he were 68 inches (172.7 cm.) tall, the volume of the body would occupy $^{4050}/_{68^3} = {}^{4050}/_{314432} = 1.288$ per cent of the cube of his height.

In the human body from infancy to adult age, this percentage of the space equal to the cube of the height of the person changes. In infants it is approximately 2.477 per cent. The relative cross-section in the infant would be twice as great as in the adult.

> By correlating this difference in ratio with the difference in body form, we are enabled to use the ratio as an index of specific differences in form in so far as the correlation holds. Since individuals do not develop uniformly and vary in body form at all stages of development, the correlation of the ratio above mentioned with body form can be at best merely approximate, but with its limitations it is of great value as an expression of build.

For convenience, Bardeen divides the weight directly by the cube of the height rather than by estimating the volume from the weight. "Thus a man 68 inches tall, weighing 150 pounds, would have a height-weight index of $^{150}/_{314432}$, or 0.000477, which is $1/27$ of the volume-cube height ratio—0.01288."

For our analysis we have taken Bardeen's chart of the typical height-weight index growth curves, recalculating all values for weight and height from the English to the metric system in which our measurements have been made This obviously brings about a change in the numerical

value of the index, the centimeter-gram index having 2.768 per cent of the value of the inch-pound index used by Bardeen.[8]

Thus we have a chart on which the height and weight of any patient irrespective of age may be plotted. We can obtain from such a curve an indication of whether a person is too heavy or too light for a given height. We can also learn whether his growth is proportional to normal growth depending on whether his curve tends to deviate from, approach, or remain parallel to the normal curve of growth.

2. *The Percentage Variation Chart.*—In making this type of chart, we have assumed a straight line to be the curves of normal height and weight as given, for instance, in the Stratz table which agrees closely with the average values of the material gathered by the Children's Bureau, U. S. Department of Labor.[9] The percentage variations from this normal curve in height and weight may be plotted chronologically on this chart, the abscissas denoting time. Thus, if an individual is shown to be greatly overweight and overheight, he may still be of normal proportions although he is too heavy and tall for his age, and would be classed as overgrown. Since these two curves are rarely identical in their deviation from the normal, and since the measurements from which they have been constructed were grouped separately on a chronologic basis, it is much more difficult to interpret them in their relationship to one another than in the Bardeen curve, because Bardeen, in constructing his curve for normal growth, makes his groupings on a basis of weight for a given height, only roughly correlating age. Furthermore, when measurements of body proportions are taken, there is less variability when they are made from groups divided on the basis of stature than when the groups are divided on the basis of age.[10] For example, a comparison of the curves 29 A and 29 B (charts 3 and 4) shows that the person may vary from the normal in both height and weight at a given age, but the relation of his height to weight as an expression of body build independent of age is not brought out so clearly by the percentage curve as by the Bardeen curve. (With both types of curves, variations in height are always in less contrast than are variations in weight, for height once attained is not lost. In other words, with gain or loss in weight three dimensions are involved; with height, only one dimension is involved.)

AVERAGES AND DEVIATIONS

In making an average curve such as the Stratz curve, a method known as smoothing is usually employed. The differences between the successive averages, indicating monthly growth, are first smoothed by substituting for each figure a

8. Bardeen (footnote 2, p. 490).

9. Statures and Weights of Children under Six Years of Age, U. S. Dept. of Labor, Children's Bureau, Bureau Publication 87.

10. Bardeen (footnote 2, p. 503).

new one equal to the average of five values—the figure itself, the two preceding, and the two following figures. The process is then repeated a second time on this smoothed series. These figures are then adjusted so that the sum of the smoothed is equal to the sum of the original figures indicating growth. These final smoothed figures are then used to construct the smoothed series of averages. The first two and the last two averages are kept unchanged.[11]

In the interpretation of these curves, the question of the extent of normal variability from the average at any point in the curve must be considered. There are two principle methods of measuring variability, namely, the standard deviation, which is an absolute value, and the coefficient of variation, which is a relative value. The first is an actual measure of how close the figures for individual cases are to the average. The second is a relative measure found by dividing the standard deviation by the average height or weight, and expressing the result by a percentage. The standard deviation affords a criterion for testing the individual case in comparison with the average.

The individual cases arrange themselves about their average in agreement with the normal gaussian equation.[12]

$$y = \frac{N}{\sqrt{2\pi\sigma}} \cdot e \frac{-x^2}{2\sigma^2}$$

when N = the number of cases.
$\pi = 3.1416$
σ = the standard deviation
$e = 2.7182818$

The measurement of variability of these cases about their average is given by σ (standard deviation). The limits $\pm 2\sigma$ give the boundary lines about the average, within which 96 per cent of the cases fall by random selection and without which 4 per cent of the cases occur. Individual cases falling without these boundaries are commonly considered to vary significantly from the normal standard.

Although it is realized that this method of approach would be most sound, the cases are too few to warrant the refinement of the biometric analysis. Consequently, we have set an artificial boundary of variability, that is $\pm$ 12 per cent from average values, which will give a sufficiently accurate estimation in view of the material itself.

No measure of variation of the height-weight indexes in the Bardeen charts has been attempted. The plotting of the curve of an individual which falls entirely on one side of the average curve is considered to have certain significance, although no simple quantitative estimation is possible.

THEORETICAL CONSIDERATION OF FOOD REQUIREMENT OF NORMAL CHILDREN

There is greater paucity of information on this subject than on the question of growth in children. We have chosen as a standard of comparison the data of Holt and Fales[13] (table 2). It is of interest to compare the diets of our diabetic children with the requirements of Holt and Fales for various ages. Such a comparison is open to criticism, for food demands must vary with body mass and activity, and not neces-

11. U. S. Dept. of Labor (footnote 9, p. 15).

12. Pearl, Raymond: Introduction to Medical Biometry and Statistics, Philadelphia, W. B. Saunders Company, 1923, pp. 243.

13. Holt, L. Emmett, and Fales, Helen L.: The Food Requirements of Children, Am. J. Dis. Child. **21**:1-28 (Jan.) 1921; **22**:370-380 (Oct.) 1921; **23**:471-480 (June) 1922; **24**:44-55 (July) 1922; **24**:311-319 (Oct.) 1922.

sarily according to age. We have made a note on the proportion of the total calories yielded by carbohydrates, protein and fat.

THE ANALYSIS OF THE GROWTH CURVES

The preinsulin course in the majority of our cases of juvenile diabetes may be described as follows: Whether overweight or underweight at the onset of the disease, there is loss of weight due to glycosuria and the restriction of diet. With the control of glycosuria, there is a disappearance of the symptoms of the disease. Ability to utilize food returns in varying degrees. The diet may be increased without the occurrence of glycosuria, and the patient may continue to grow in

TABLE 2.—*Holt and Fales' Standards for Normal Children, With Which We Have Arbitrarily Compared "Best Growth" Diets of Diabetic Children*

	Boys				Girls			
Age	Calories, Daily	Fat, Gm. Daily	Carbohydrate, Gm. Daily	Protein, Gm. Daily	Calories, Daily	Fat, Gm. Daily	Carbohydrate, Gm. Daily	Protein, Gm. Daily
1	950	35.8	115.8	35.0	940	35.0	114.7	34.5
2	1,135	42.8	138.3	41.5	1,110	41.8	135.3	40.5
3	1,275	48.0	155.5	47.0	1,230	46.4	150.0	45.0
4	1,380	52.0	168.3	50.5	1,300	49.0	158.5	47.5
5	1,490	56.2	181.8	54.5	1,410	53.2	172.0	52.0
6	1,600	60.3	195.2	58.5	1,520	57.3	184.5	56.0
7	1,745	65.8	212.8	64.0	1,660	62.6	202.2	61.0
8	1,920	72.5	234.2	70.5	1,815	68.5	221.3	63.5
9	2,110	80.0	257.5	78.0	1,990	75.0	242.8	73.0
10	2,330	87.7	284.0	85.5	2,195	82.5	268.0	80.0
11	2,510	94.5	306.0	92.0	2,520	95.0	309.8	92.5
12	2,735	103.0	333.5	100.0	2,865	108.0	349.5	105.0
13	3,040	114.5	371.0	111.0	3,210	121.0	391.7	118.0
14	3,400	128.0	414.8	125.0	3,330	125.5	406.0	122.0
15	3,855	145.0	470.0	142.0	3,235	122.5	394.5	119.0
16	4,090	154.0	499.0	150.0	3,160	119.0	385.2	116.0
17	3,945	148.5	481.5	145.0	3,060	115.5	373.2	112.0
18	3,730	140.5	455.0	137.0	2,950	111.0	330.0	108.0
Adult	3,265	123.0	400.0	120.0	2,640	100.0	322.0	97.0

height and, usually, to regain some of the weight lost. This continues until the glycosuria again begins, followed very soon by new loss of weight. Possibly if the diet is severely restricted, no gain but a constant loss in weight results, with probably little or no gain in height.

The return of glycosuria may be due solely to the inevitable loss in tolerance observed in all our cases of diabetes mellitus, and always hastened in its course by a break in diet. Children are notoriously difficult to treat because of their lack of understanding of the necessity for the control of food intake in the face of the instinctive urge to take food, the stimulus of appetite, the taste for good things, and the many and frequent temptations which seem inevitable in the life of the child. A diabetic child without glycosuria usually feels well and sees no reason why he should not do as other children.

These various periods of glycosuria with loss of weight, aglycosuria with partial increase in tolerance, gain in weight, and sometimes growth

in height, may repeat themselves until finally a state is reached in which the diet is severely restricted, the child is emaciated, his activity curtailed and all growth in height seems to have stopped. The end, then, in most cases is inevitable, due to starvation, coma or intercurrent infection. It is in this state that insulin proves a life saver.

In order to prolong life as far as possible and to prevent rapid increase in the severity of the disease, it is absolutely essential to control glycosuria by diet restriction, and this is made less difficult if the patient is kept underweight. Such a course is well illustrated by the preinsulin portion of curve 29 A (chart 3).

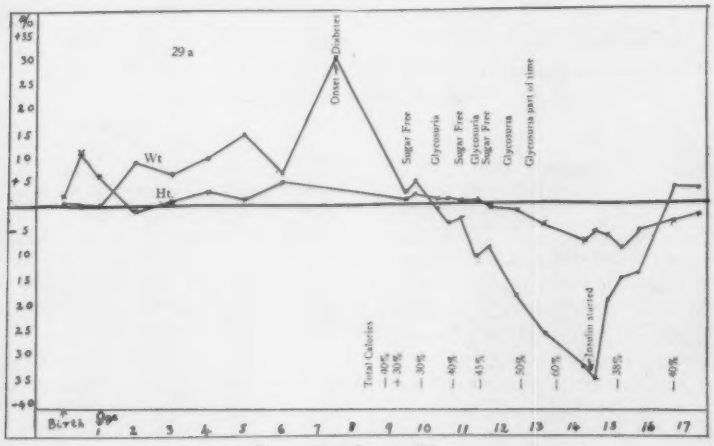

Chart 3.—Curve 29 a.

CASE RECORDS

The following ten cases well illustrate the types of curves and their significance:

CASE 29.—Curve 29 A (chart 3) is a percentage variation curve. At a given age it represents the plus or minus deviation from the straight horizontal line which is assumed to be the curves of normal height and weight according to age for both sexes. This curve shows that the child was of average height and weight for his age, and also probably too heavy for his height at the onset of diabetes. The variation is extreme. But this curve cannot give a clear idea of the true relation of weight to height, nor of the progress of growth. It does, however, show the details of the preinsulin course of the disease.

The course under insulin therapy as shown by curve 29 A (chart 3) is dramatic. It shows gain in weight and height until near normal for the given age. With the administration of insulin, there occurred early in the fifteenth year a halt in increase in height and weight, while during this time the patient often showed heavy glycosuria. After this period, in order to keep the patient sugar-free, it was necessary to more than double the insulin dose.

When we turn to curve 29 B (chart 4), plotted according to Bardeen, we find that the proportional growth of the child was remarkably close to normal. When he was 1 year old he had reached the approximate stature of a child of

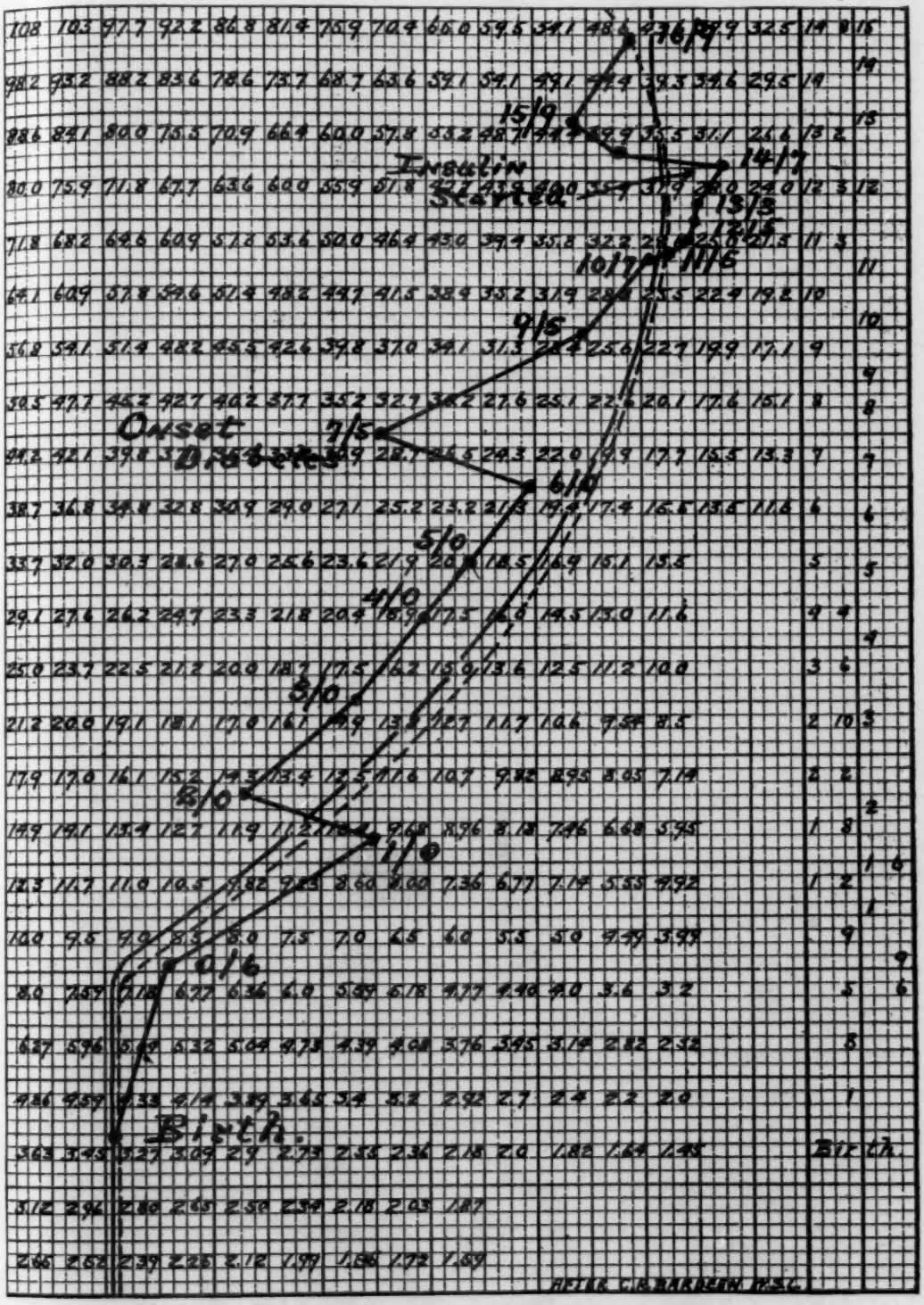

Chart 4.—Curve 29 B.

1½ years. Then a rapid and unexplained gain in weight occurred. For the following four years, the child grew closely to normal and in fair proportion, but was always too heavy for his height. During his sixth year he again made a rapid gain in weight. Most of this gain occurred during convalescence from measles, and was brought to an abrupt termination by the onset of diabetes. His growth in height continued for about three years after the onset of this disease in spite of loss in weight. Then it almost ceased and at the age of 14½ years he had the approximate stature of a child of 12½, being light for his height. With insulin treatment he rapidly gained weight, and in about a year began to grow. At 16½ years of age, although his stature was approximately that of a boy of 14½ years, he grew well for the last year and a half. Although we have noted age in the discussion of this case, the significance of the Bardeen curve is clearly seen to show the relation of weight to height (body build) rather than the relation of weight or height to age. Both parents are tall, and it might be expected that the boy would be more than medium height. There is a

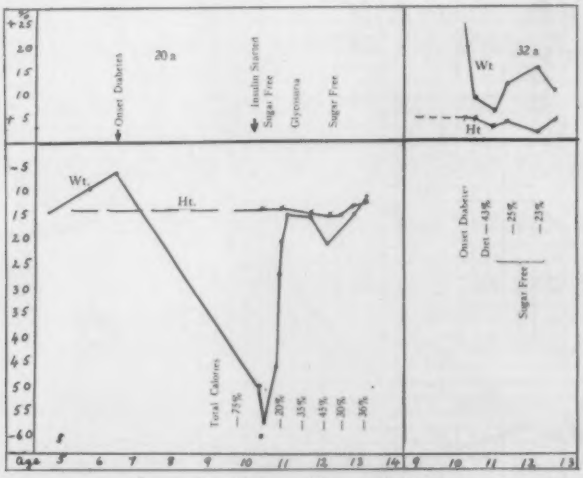

Chart 5.—Curves 20 a and 32 a.

family history of diabetes. There is also the possible etiologic factor of acute infection (measles).

We wish to point out that under insulin therapy growth in height was not resumed until the child had first gained weight to the extent of being slightly heavy for his height, that is, until he had regained the same proportions he had maintained previous to the onset of diabetes.

Case 32.—This patient did not receive insulin. So far the course of the disease has been controlled by diet. The patient was kept sugar-free and growth in height continued. He was somewhat heavy for his height. The parents were of medium build.

Case 17.—In this patient insulin therapy was begun at once. Although there was an initial loss in weight, it was of short duration, and was followed by the usual rebound or gain in weight to the point of overweight. Growth in height continued without cessation, and the Bardeen curve, 17 B, shows a practically normal development of body build. An increasing dose of insulin was necessary to keep the patient sugar-free. The only etiologic factor apparent was an excessive intake of sweets.

Case 8.—In this patient, the onset of diabetes occurred at 3 years of age. Curve 8 B (chart 6) shows the patient greatly overweight and above average

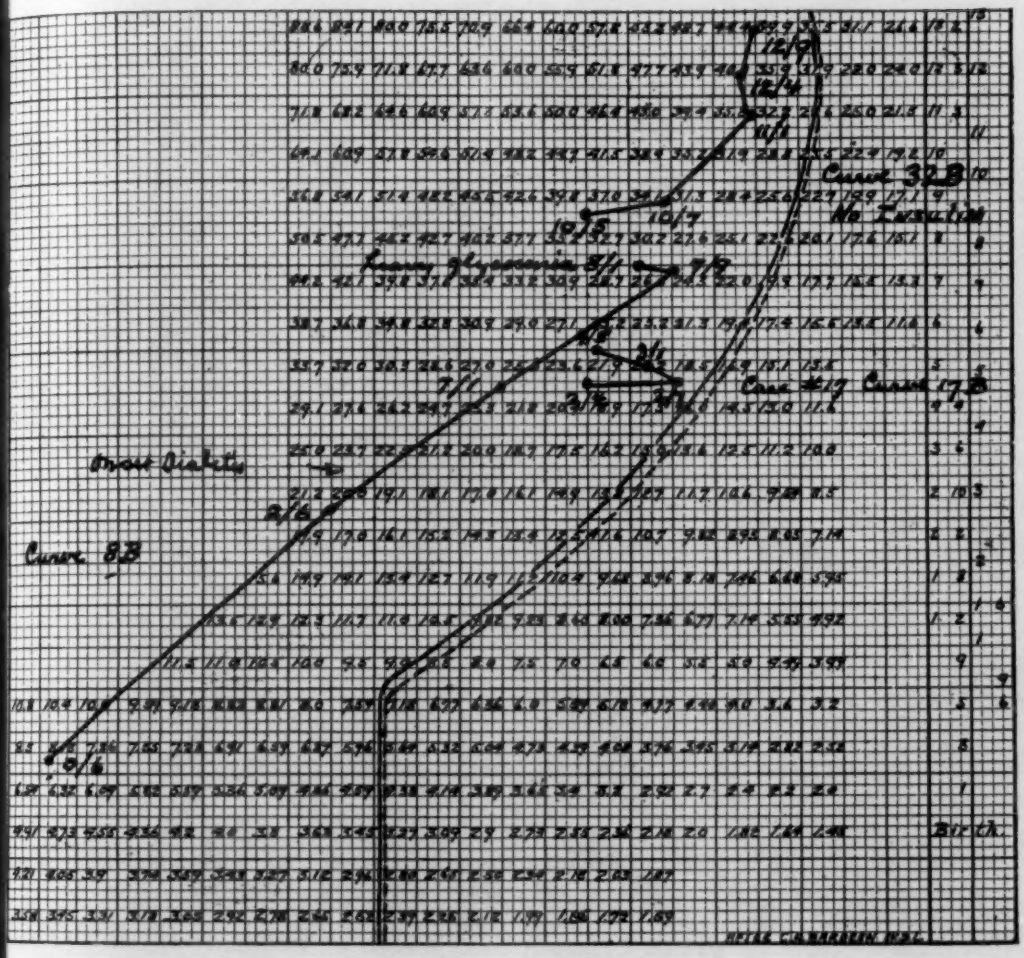

Chart 6.—Curves 8 B, 17 B and 32 B.

height for age. Subsequent loss of weight with later gain under administration of insulin is more strikingly shown in 8 B than in the Bardeen curve, since the necessary measurements for height are lacking. Overfeeding, excessive carbohydrate and family history of diabetes may all have played a rôle in the etiology. The curves illustrate the effect of heavy glycosuria on growth in

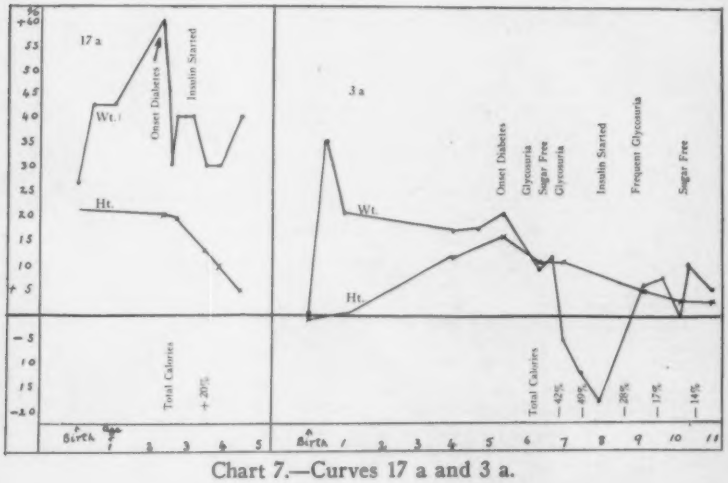

Chart 7.—Curves 17 a and 3 a.

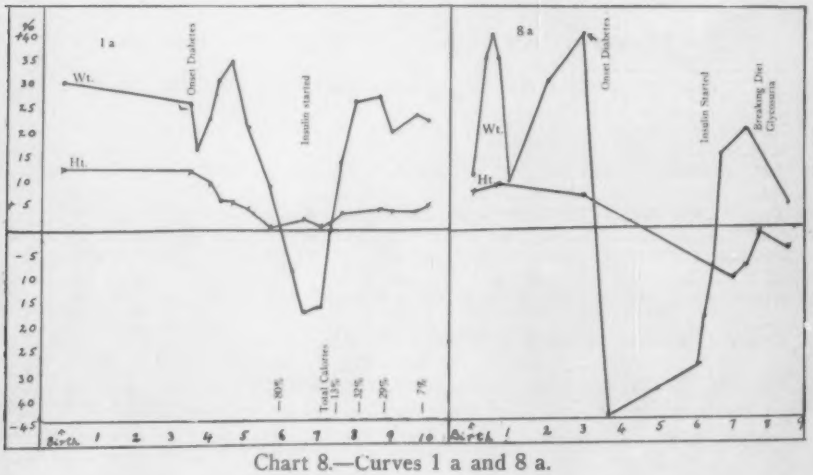

Chart 8.—Curves 1 a and 8 a.

height during the last year of the disease. During insulin therapy, diet control was disregarded, and although there was slight actual gain in weight gain in height ceased.

Case 20.—The patient was said always to have been very short. His parents were short. There was a family history of diabetes, and it was said that he was a difficult child to feed. The family forced the feeding to promote growth, but without avail. There were no other etiologic factors known. Although below height and weight, the Bardeen curve shows that he was not far from

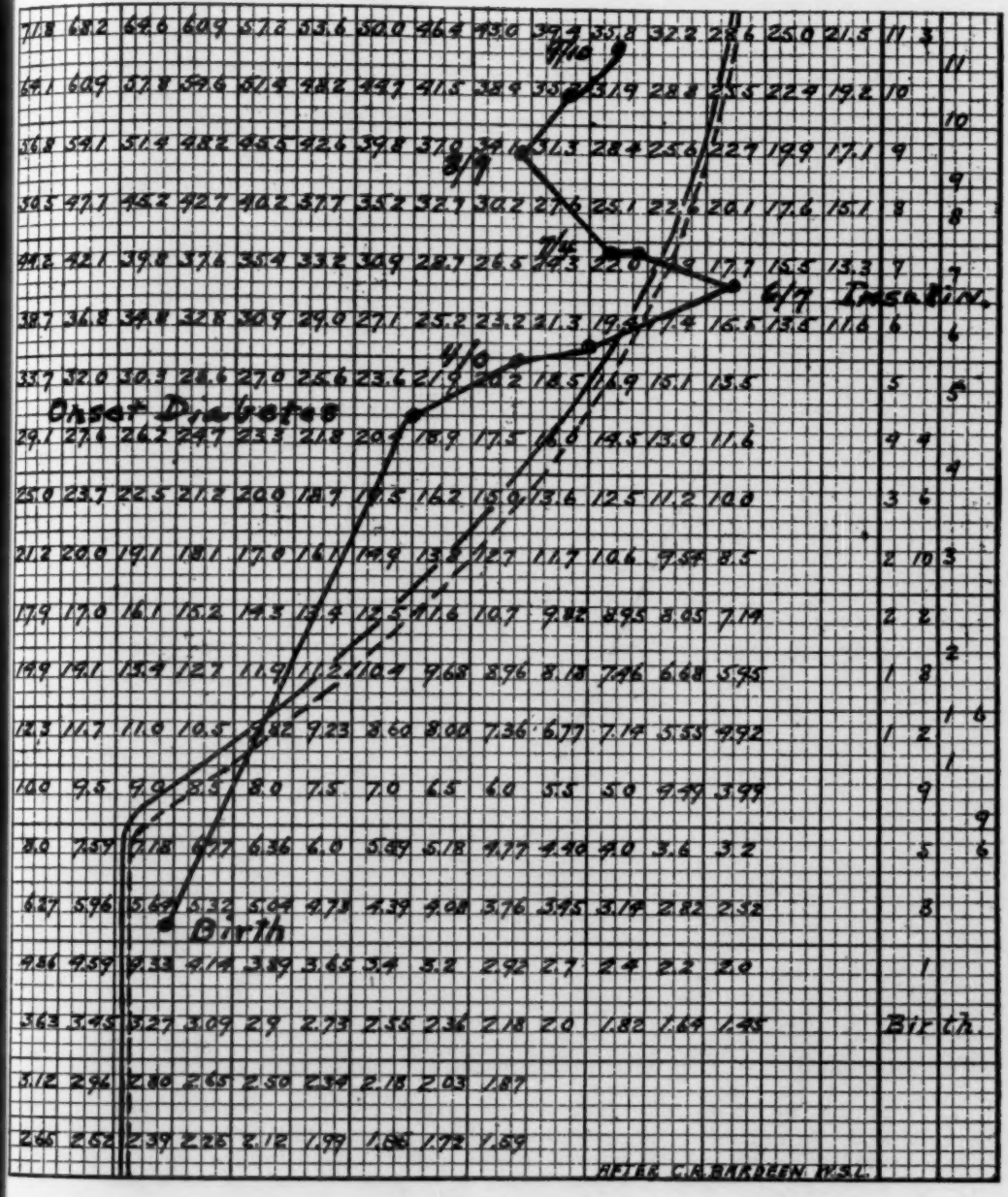

Chart 9.—Curve 1 B.

normal stature at the onset of the disease. Again we call attention to the considerable gain in weight before the resumption of growth in height and to the fact that the patient had grown better when sugar-free.

CASE 3.—At the age of 4 years, this patient had the approximate stature of a child of 5½. She grew in normal proportions until the age of 5, when she had the stature of a child of 8; then diabetes developed. Growth ceased and the patient lost weight. After insulin treatment she first gained weight until slightly overweight, when she began to grow in height. This growth during insulin treatment did not keep pace with growth in height before the onset of diabetes, but was better than during the preinsulin year. Glycosuria occurred frequently. At the age of 11 the patient had the approximate stature of an 11 year old child.

CASE 1.—This patient was overweight at the onset of diabetes, but lost steadily and was kept sugar-free on a greatly restricted diet (—80 per cent in calories). This curve shows clearly the gain in weight after insulin therapy before the resumption of growth in height. It also shows the tendency toward normal proportions, although the patient was probably short for his age. Both parents were large and tall.

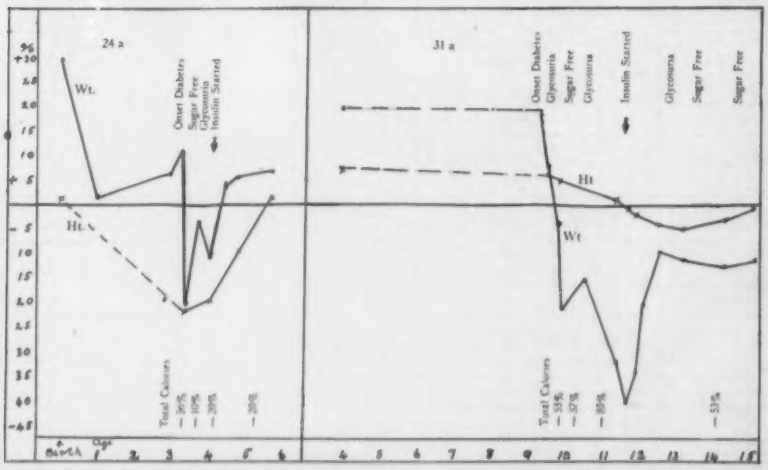

Chart 10.—No measurements were made between birth and the onset of illness, but photographs show that the child was definitely taller than the average, as compared with objects of known heights in photographs.

CASE 31.—The study of curve 31 a (chart 10) shows the patient overweight for height at the onset of diabetes. Measurements during the prediabetic life of the child are lacking, but all of a series of photographs showed a fat, rather flabby girl whose height appeared to be above the average. Her parents were both short. Soon after the onset of the disease the patient almost ceased growing, and under insulin treatment has not yet regained average height for her age although she has grown well in height during the last year. The Bardeen curve, 31 B (chart 11), shows recovery of normal proportions under insulin treatment, and that growth in height was not resumed until the patient had become somewhat heavy for height. The usual history of the preinsulin course of the disease, as was outlined under case 1, is again well illustrated.

CASE 24.—From birth this child was heavy for her height. Although the percentage variation curve, 24 a (chart 10), shows her to be near normal weight and height at 1 year, the Bardeen curve shows her greatly overweight at the onset of diabetes, with an approximate stature at 3 years of a child of 1½ years.

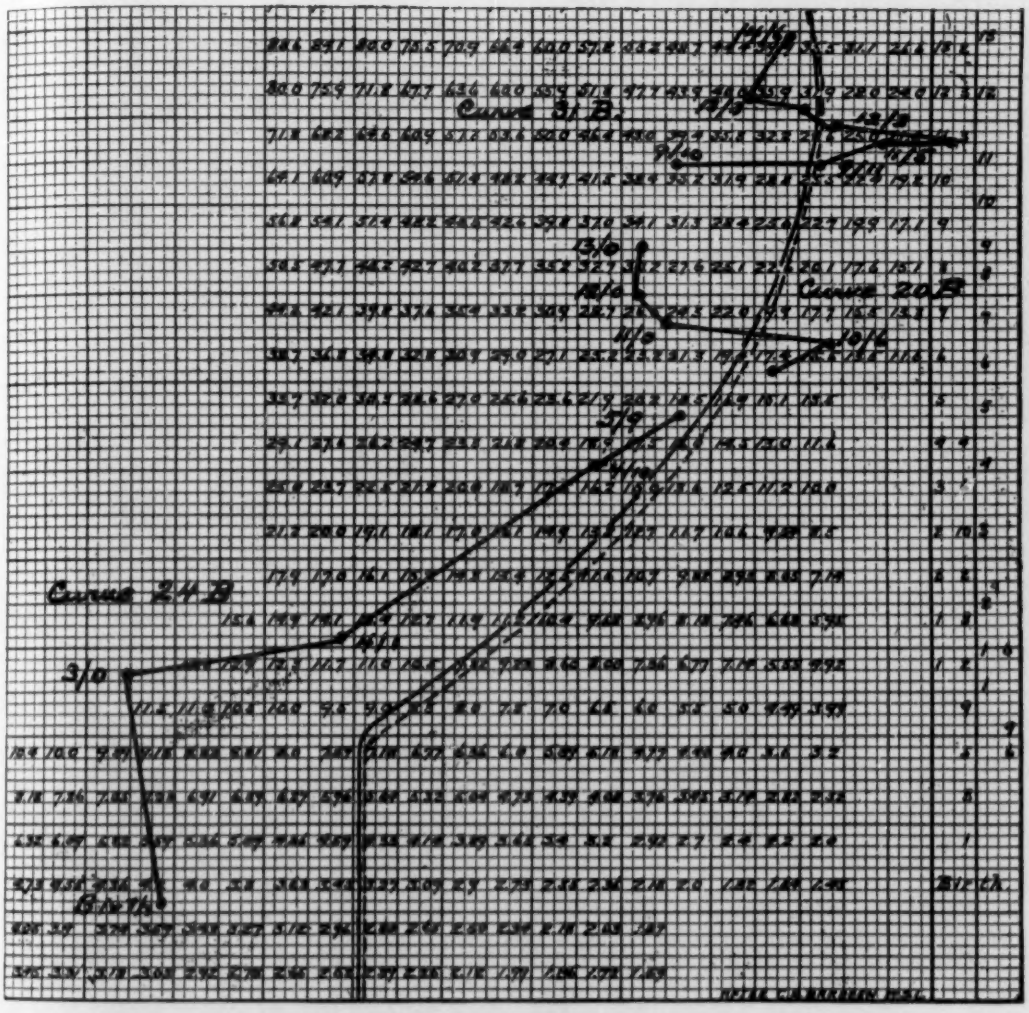

Chart 11.—Curves 31 B, 24 B and 20 B.

Between the ages of 3 and 4 her gain in stature was slight. She increased in both height and weight immediately after insulin administration was begun. The growth during the first ten months of insulin treatment was remarkable, but during the last year there have been periods of consecutive weeks when the patient showed heavy glycosuria. Gain in stature continued, but was not so rapid. At 6 years of age, she was about normal in proportion, but had the stature approximately of a girl of 5.

CASE 11.—This case was an excellent example of the difficulty of interpreting the percentage variation chart as a picture of relation between weight and height. This can be seen by a comparison of curves 11 a (chart 12) and 11 B (chart 13). At the time of writing, the percentage variation of curve 11 a showed the patient to be 37 per cent overweight and 12 per cent overheight. He did not look fat, but was well nourished. He was active in athletics. His diet was low in total calories, and he was kept sugar-free. When plotted on the Bardeen chart, curve 11 B (chart 13) showed a constant tendency to arrive at

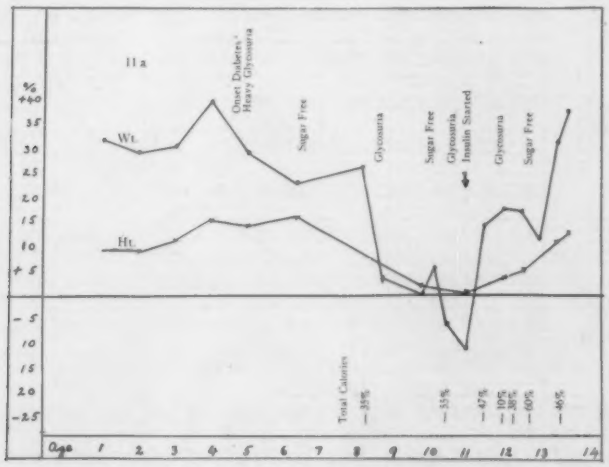

Chart 12.—Curve 11 a.

normal proportions. The boy was overgrown, but apparently of average weight for height. At the age of 14, he had the approximate stature of 16½ years. This case also illustrates the preinsulin course of diabetes, especially the relation of glycosuria to downward progress of the disease. Moreover, it shows the effect of glycosuria in relation to growth under insulin treatment.

COMMENT

A study of the growth curves of the thirty-four cases of diabetes in children and one case of possible renal diabetes when plotted on the percentage variation chart shows that at the onset of the disease six were underweight for age and twenty-nine overweight.

However, when these two groups were plotted on the Bardeen chart, of the six underweight for age (table 3) three were overweight for height, two were of normal proportions and one was light in weight for height. Of the twenty-nine overweight for age (table 4), one was of normal proportions and about the average height for age. Nine

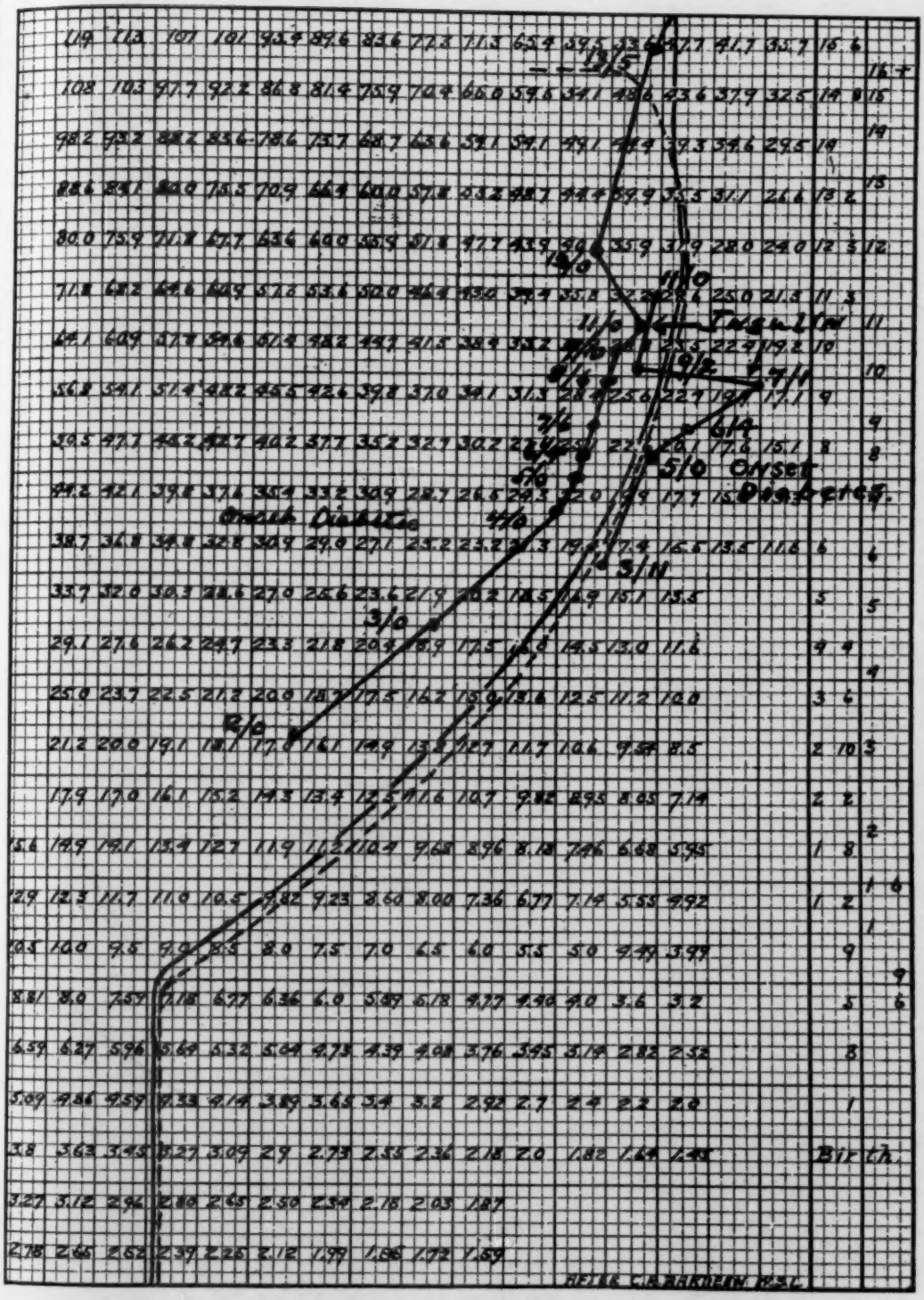

Chart 13.—Curves 3 B and 11 B.

TABLE 3.—*Children Underweight for Age According to Percentage Variation Curves*

Case	Overfed, Calories and Carbohydrates	Family History, Diabetes	Onset Post Acute Infection	Nationality	Stature Parents*	Remarks on Weight for Height as Plotted on Bardeen Chart
2	Yes	No	No	American	T. T.	Heavy for height
23	Yes	Yes	No	Jewish	M. M.	Heavy for height
7	No	Yes	No	American	?	Heavy for height
9	Yes	No	Pneumonia	Colored	S. S.	Normal proportions
15	No	No	No	Welsh	T. M.	Normal proportions; short for age
20	Yes	Yes	No	Jewish	S. S.	Light for height

* Stature of parents is approximate only; estimation of body build not made.

TABLE 4.—*Children Overweight for Age According to Percentage Variation Curves*

Case	Overfed, Calories and Carbohydrates	Family History, Diabetes	Onset Post Acute Infection	Nationality	Stature Parents*	Remarks on Weight for Height as Plotted on Bardeen Chart
				Boys		
1	Yes	No	No	Irish	T. T.	Heavy for height; at 3½ years, stature 4½ years
4	Yes	No	No	Jewish	M. M.	Heavy for height; at 14 years, stature 15½ years
5	Yes	Yes	No	American	T. S.	Normal proportions; at 15 years, stature 17 years
6	No	No	Whooping cough	American	M. M.	Heavy for height
10	Yes	No	No	Jewish	S. S.	Heavy for height
11	Yes	No	No	American	T. M.	Heavy for height; at 13 years, stature 15 years
12	Yes	No	No	Swedish	T. T.	Heavy for height; at 9 years, stature 12 years
13	Yes	Yes	No	Jewish	M. M.	Heavy for height; at 3 years, stature 5½ years
14	Yes	No	No	American	M. M.	Normal proportions; at 6½ years, stature 8 years
16	Yes	Yes	No	Jewish	S. S.	Heavy for height; at 11½ years, stature 14 years
19	Yes	No	No	American	T. T.	Normal proportions; at 11 years, stature 12½ years
26	Yes	No	No	Jewish	M. M.	Heavy for height
27	Yes	No	No	Jewish	S. S.	Heavy for height
28	Yes	Yes	Measles	American	S. M.	Heavy for height; at 10¾ years,
29	Yes	Yes	Measles	American	T. T.	Heavy for height; short for age
32	Yes	No	No	American	M. M.	Heavy for height stature 12 years
33	Yes	Yes	No	Jewish	M. M.	Heavy for height; at 9½ years, stature 11½ years
34	Yes	No	No	American	T. T.	Heavy for height; at 8 years, stature 10 years
35	Yes	No	No	American	M. M.	Heavy for height
				Girls		
3	Yes	No	No	American	?	Normal proportions; at 4 years, stature 6 years
8	Yes	Yes	No	Spanish	M. M.	Heavy for height
17	Yes	No	No	Jewish	M. M.	Heavy for height; at 2½ years, stature 4½ years
18	Yes	Yes	No	German	S. S.	Heavy for height
21	Yes	Yes	Otitis	Jewish	S. S.	Heavy for height
22	No	No	No	American	M. S.	Heavy for height
24	Yes	No	No	Hungarian Jew	M. M.	Heavy for height
25	Yes	No	Scarlet	Jewish	M. S.	Normal proportions
30	Yes	No	No	German	T. T.	Heavy for height; at 7½ years, stature 9 years
31	Yes	Yes	No	Dutch	S. S.	Heavy for height; at 9½ years, stature 10½ years

* Stature of parents is approximate only; estimation of body build not made.

were overweight for height, six being about the average height for age and three shorter than average for age. Four were of normal proportions and taller than average for age. Twelve were heavy for height and taller than average for age.

Thus in sixteen of the thirty-five cases the growth stimulus or impulse was well above average and the child was, or still is, large for his age.

We can say, therefore, that in thirty-one of these thirty-five cases of diabetes, excess weight for height or excess height, or both, is present. In two of the seven children of normal proportions the onset followed acute infections. In four there was no etiologic factor except probably overfeeding. In one there was a family history of diabetes. In the one child light in weight for height and not overgrown, there was a long history of forced overfeeding, plus Jewish race and a family history of diabetes. There was, then, in these diabetic children before the onset of the disease, at the time of onset and even after onset when on insulin dosage, a definite deviation of the growth impulse from normal and a tendency to overweight.

It seems, then, from an analysis of the cases that with the onset of diabetes when insulin is not used in therapy, the growth process slows down or ceases altogether. To have growth in height continue at normal rates, even in moderate diabetes, is exceptional but has occurred, as, for example, in cases 21 and 32, when growth in height has continued normally for well over a year without the administration of insulin. However, growth in height decreases as the disease progresses.

Insulin is the agent which enables the body to utilize carbohydrate and obtain full benefit from protein and fat. Whether it has other action which may be analogous to a vitamin or hormone as special stimulant to growth, we do not know, but it is not necessary to assume any such action.

The effect of insulin on growth in these severe cases of diabetes may be noted as follows:

1. There is immediate gain in weight.

2. There is usually coincident gain in strength and often improvement in disposition in spite of the inconvenience of insulin injections.

3. Gain in stature usually follows gain in weight. Some of these children have begun to grow tall within a month; in a few growth did not begin for more than a year. Growth in height does not usually begin until the patient has attained normal weight for height, and in some cases not until the patients become heavy for height. In these last cases there is no resumption of growth in height until the patient has regained the relative body build he had at the onset of diabetes. As time goes on and stature increases, these patients tend to assume normal proportions in most instances. This may be the result of controlled diet.

4. The gain in stature occurs sooner when the patient is kept sugar-free.

5. When there is heavy glycosuria for a long time, that is, for months, there sometimes is failure of growth in stature. This may be simply a matter of caloric loss through the urine, although we have observed growth to stop when gain in weight is present.

6. When irregularity in insulin dosage or in food intake, or both, is practiced it soon leads to glycosuria. The effect is much the same as when in the preinsulin period there occurred a break in diet with its consequent glycosuria. That is, the "tolerance" is lowered and the efficiency of the insulin seemingly decreased. This glycosuria, if of long standing, tends to bring about a state in which disproportionately large doses of insulin in relation to carbohydrate intake are needed to make the patient sugar-free. There also occurs an instability of the mechanism controlling blood sugar so that a patient quickly oscillates from glycosuria to shock. Stability is regained only after a long period of regular and, usually, low diet. During this period of instability, it is often impossible to keep the patient sugar-free throughout the twenty-four hours without several doses of insulin.

Is there an optimum weight at which to keep the diabetic child? The answer is, "yes." It is probably the normal average weight for height as given in Bardeen's tables. Certainly the child should not for any long period be allowed to remain overweight for height, if one bears in mind that the deviation from the normal curve may vary $\pm$ 12 per cent.

FOOD INTAKE IN RELATION TO GROWTH CURVE

To plot all the changes in diet during the courses of these cases of diabetes would be exceedingly tedious, and it is doubtful whether the information obtained would be valuable, for in many cases there were numerous changes of diets for short periods only. On the growth charts for percentage variation we have indicated the approximate deficiency of total calories as compared with the Holt and Fales [18] standard for age. These show that children gained in weight and sometimes in height on diets low in total calories.

In order to obtain more accurate knowledge of the relation of food intake to growth, we have prepared table 5.

Many of the patients were treated by restricted diet alone before we had insulin as a therapeutic agent. In the first column we have given the total caloric deficit as compared with the standards of Holt and Fales for age, in order to show the amount of diet restriction that was necessary to keep these patients sugar-free during the most severe part of their preinsulin course. The second column gives the approximate quantity of protein in grams per kilogram of body weight during the same

period. The third column gives the caloric deficit of the actual diet during the period of best growth. The figures in columns 3, 5, 6 and 7 indicate the approximate diet in all cases, and for most of these cases the figures represent accurate amounts of food given over a period of several months. The caloric deficiency as noted does not include calories lost through carbohydrate in the urine. In all but four cases insulin was used. During these periods of best growth, the patients were usually

TABLE 5.—*Food Intake*

Case	Caloric Deficit, Preinsulin Course, per Cent	Protein, Gm. per Kg.	Caloric Deficit, Best Growth, per Cent	Protein, Gm. per Kg.	Percentage of Total Calories During Period of Best Growth			Insulin, Dose	Weight of Patient, Kg.
					Protein	Fat	Carbohydrate		
				Boys					
1	—80	1.5	— 7	2.0	14.6	47.4	38.0	70	34
4	—88	0.5	—48	4.5	15.4	65.4	19.2	40	52
5	—42	0.78	—14	1.2	13.1	57.8	29.1	76	74
6	—50	3.0	± 0	2.9	16.5	48.1	35.4	5	24
7	—20	1.5	—42	1.6	14.0	59.7	26.3	52	50
9	± 0	3.5	± 0	3.7	16.4	56.3	27.3	47	16
10	—33	1.0	—58*	1.0	8.3	86.0	5.7	0	20
11	—45	2.0	—50	2.0	20.8	40.5	38.7	52	45
12	—25	1.7	—29	1.6	18.6	50.4	31.0	35	46
13	± 0	4.0	†	3.2	....	....	....	0	..
14	—65	2.0	—47*	1.5	13.3	80.0	6.6	0	20
15	—60	0.75	—29	2.3	13.2	68.0	18.8	40	30
16	—80	1.0	—31	1.6	14.0	73.6	12.4	65	55
19	— 4	2.0	—33	1.6	12.1	66.0	21.9	10	31
20	—80	2.5	—33	2.4	15.2	48.2	36.6	78	31
26	—50	1.7	—47	1.4	15.9	54.2	29.9	50	58
27	—36	4.0	± 0	3.4	13.0	74.0	13.0	10	11
28	—40	2.5	—40	1.9	17.2	38.1	44.7	43	39
29	—60	3.5	—41	1.9	14.5	65.6	19.9	55	43
32	—40	1.8	—23	2.2	17.5	57.2	25.3	0	41
33	—70	1.5	—47	1.0	15.5	35.1	49.5	115	48
34	—33	?	— 9	2.6	17.5	53.2	29.3	17	29
35	—33	2.2	— 7	2.6	17.0	52.0	31.0	38	29
				Girls					
2	—81	2.5	— 6	3.7	18.5	49.5	32.0	41	15
3	—60	2.3	—14	2.2	13.4	51.6	35.0	75	31
8	— 8	2.5	—43	1.7	17.8	62.5	19.7	38	26
17	—10	2.3	+ 1	2.5	17.1	53.2	29.7	38	22
18	—70	2.6	—49	1.3	21.3	48.3	30.4	80	53
21	—55	1.5	—40	1.3	‡	....	....	70	55
22	—87	3.2	—34	2.3	16.9	67.3	15.8	50	36
23	—10	3.0	± 0*	3.8	18.5	63.0	18.5	21	16
24	—30	2.4	— 8	2.2	12.7	65.0	22.3	40	18
25	—80	1.8	—42	2.3	20.8	47.2	32.0	43	29
30	—11	2.7	—10	2.0	15.2	34.3	50.5	50	27
31	—85	1.0	—48	2.0	19.0	64.4	16.6	60	39

* No growth.
† Normal growth. Renal diabetes. Heavy glycosuria.
‡ Normal growth irrespective of diet.

sugar-free. When glycosuria occurred in appreciable quantities, it was checked as quickly as possible. During short periods, because of a break in diet or acute respiratory infection, four of the patients showed heavy glycosuria and may have lost as much as 20 per cent of the calories in the urine. Quantitative data are lacking in some of these instances. In the columns 8 and 9 we have noted the insulin dosage and the mean weight for the period.

A survey of these cases shows that with insulin treatment growth took place on diets often from 40 to 50 per cent deficient in total calories, as compared with Holt and Fales' standard.

The percentage of the total calories derived from the protein varied in the boys from 12.1 to 20.8 per cent, with an average of 15.5. In girls the protein quota of the total calories varied from 12.7 to 20.8 per cent, the average being 17.7 per cent. Although the total calories were greatly deficient, the protein quota is close to the Holt and Fales standard of 15 per cent. The grams of protein per kilogram of body weight run much under the Holt and Fales standard except when the patient's body mass is small as in cases 2, 9, 23 and 27. If we omit these four cases, the grams per kilogram of protein averages about 1.9.

The proportion of the total calories from fat (50 per cent) and carbohydrate (35 per cent) are roughly the opposite of the Holt and Fales standards (50 per cent carbohydrate, 35 per cent fat), although cases 28, 30 and 33 are in accord with the Holt and Fales proportions.

For the last two years we have purposely prescribed 50 per cent of total calories from carbohydrate, and 35 per cent from fat in some of the dietaries with most satisfactory results. The child is happier because of the more liberal carbohydrate diet which admits a greater variety with less temptation to food theft. This type of diet has been used without an increase of insulin in proportion to the carbohydrate increase, and occasionally a surprising decrease in the dose of insulin.[14] In such cases, however, there has not usually been an increase in the total calories.

The cases show, then, that a diabetic child will grow well when he is kept sugar-free with sufficient insulin and receives a protein ration of about 1.5 to 2 Gm. per kilogram of body weight, although the total calories in the diet are only from 50 to 70 per cent of the Holt and Fales standard. Good growth has occurred on 0.8 Gm. protein per kilogram of body weight.

The substances which enable growth to take place, namely, the vitamins and salts, have been present in adequate amounts in all of the diets, owing to the quantity of fresh vegetables, milk, butter, and fresh animal and vegetable proteins used.

The amount of food, then, as represented by total calories need not equal the normal standard of Holt and Fales, nor need the proportions of protein, fat and carbohydrate correspond to those of the normal diet of Holt and Fales. We suggest, however, that a proportion of carbohydrate relatively higher than fat may possibly be used to advantage. But insulin is the essential factor in regaining normal growth in height and weight.

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14. Geyelin, H. Rawle: Recent Studies of Diabetes in Children, Atlantic M. J. **29**:825 (Sept.) 1926.

The criterion for sufficient total calories is an observed increase in growth plus, if necessary, determinations of the nitrogen balance over a sufficient period to establish the fact that it is positive.

Is there, then, an optimum amount of food for a given child? The answer is not as clear as in the case of weight, since food supply must vary with activity. However, these cases show that diabetic children may lead "normally active" lives, and attain average stature and strength on an amount of food which, expressed in total calories, is about two-thirds the Holt and Fales standard of normal requirement.

A protein quota of 15 per cent of the total calories is probably ample. It has not been possible in many of our cases to increase the carbohydrate calories to 50 per cent of the total. Most of our group of cases have received 50 per cent of their total calories from fat, and but 35 per cent from carbohydrate. However, as we have gained experience with insulin therapy, we have been able to increase the number of calories from carbohydrate with apparent benefit. This is especially true in older children with greater body mass. In children with small body mass there is a tendency to an unstable balance between food intake and insulin dosage. Rapid oscillation between hypoglycemia and hyperglycemia seems to take place more easily. This may be because of the difficulty in keeping the food intake constant in such children even under ward control and careful watching. In the care of such patients at home, unless the service of a special nurse can be had, stealing food, irregular hours for meals, extreme variability in the amount of exercise, and ordinary dietary upsets are difficult to avoid.

The course of patients when there is absolute regularity of the quantity of food and hours of meals, with a similar regularity for the time and the dose of insulin, is extremely satisfactory, provided there are no untoward complications in the disease.

THE BASAL METABOLIC RATE

In view of the fact that the use of insulin permits a diet relatively low in total calories, we have prepared table 6 showing the basal rates whenever available. When the observation was not made at the time of best growth, we have calculated the surface area from height and weight, using Boothby and Sandiford's [15] chart, and from those values, using Talbot's [16] figures for calories per square meter referred to age, have calculated the normal basal rate. The diet received during the period of best growth has been noted as a percentage of the actual or estimated basal rate.

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15. Boothby, W. M., and Sandiford, R. B.: Nomographic Charts for Calculation of Metabolic Rate by Gasometer Method, Boston M. & S. J. **185**:337 (Sept. 22) 1921.

16. Talbot, F. B.: Standards of Basal Metabolism in Normal Infants and Children, Am. J. Dis. Child. **21**:519 (June) 1921.

TABLE 6.—*Metabolic Rates*

Case	Years of Age	Observed Basal Rate	Body Surface in Square Meters	Estimated Basal Calories per 24 Hours	Total Calories of Diet, Percentage	Remarks
			Boys			
1	6	—18	0.75	810	.....	Preinsulin
	6	—13	0.80	864	.....	Preinsulin
	6	— 6	0.83	896	.....	Insulin for 2 weeks
	9	None	1.10	1,122	Basal +74	Insulin; best growth
4	15	—28	1.6	1,550	.....	
	16	None	1.73	1,610	Basal +43	Insulin; best growth
5	15	None	1.80	1,706	Basal +58	Insulin; best growth
6	7	None	0.9	954	Basal +82	Insulin; best growth
7	14	None	1.47	1,390	Basal +68	Insulin; best growth
9	5	—13	0.64	704	.....	
	6	None	0.73	786	Basal +90	Insulin; best growth
10	10	—16	0.84	850	Basal +15	No insulin; no growth
	10	— 4	0.85	800	.....	
11	11	—11	1.10	1,100	.....	Insulin 4 months
	12	—14	1.20	1,300	.....	Insulin
	12	None	1.25	1,355	Basal + 2	Insulin; best growth
12	9	—19	1.27	1,295	.....	No insulin
	10	— 3	1.38	1,394	Basal +19	Insulin; best growth
13	4	None	0.81	909	Basal +200	Normal; renal diabetes
14	7	None	0.83	1,872	Basal —50	No insulin; no growth
15	13	— 9	0.98	940	Basal +75	No insulin; best growth
16	14	— 6	1.20	1,226	.....	No insulin
	14	—30	1.33	1,262	.....	No insulin
	15	None	1.56	1,480	Basal +79	Insulin; best growth
19	11	—32	1.15	1,150	Basal +47	No insulin; no growth
20	10	—15	0.6	607	.....	No insulin
	10	+ 6	0.65	657	.....	Insulin
	10	+ 5	0.83	840	.....	Insulin
	11	+ 7	0.88	888	.....	Insulin
	13	None	1.01	975	Basal +107	Insulin; best growth
26	15	—25	1.43	1,335	.....	No insulin
	17	None	1.67	1,587	Basal +35	Insulin; best growth
27	1½	....	....	.....	.....	No data
28	13	None	1.80	1,240	Basal +48	Insulin; best growth
29	14	+ 2	1.08	960	.....	Insulin
	14	— 3	1.05	1,000	.....	Insulin
	15	None	1.32	1,235	Basal +83	Insulin; best growth
32	12	None	1.5	1,272	Basal +66	Insulin; best growth
33	10	— 8	1.15	1,162	.....	Insulin; best growth
	11	None	1.41	1,410	Basal — 8	Insulin; best growth
34	8	None	1.04	1,082	Basal +61	Insulin; best growth
35	8	None	1.0	1,040	Basal +72	Insulin; best growth
			Girls			
2	4	None	0.61	620	Basal +96	Insulin; best growth
3	11	None	1.09	1,160	Basal +46	Insulin; growth fair
8	7	None	0.90	972	Basal + 7	Insulin; best growth
17	4	None	0.62	620	Basal +112	Insulin; best growth
18	20	+ 2	1.35	1,175	.....	Insulin just started
	20	— 4	1.33	1,175	.....	
	22	None	1.47	1,277	Basal + 6	Growth occurred through 22
21	16	—14	1.51	1,312	.....	
	17	—12	1.54	1,338	Basal +27	No insulin; no growth
	18	None	1.57	1,363	Basal +61	Grew irrespective of diet
22	14	+ 5	1.0	980	.....	No insulin
	14	— 9	0.99	950	.....	No insulin
	15	—26	0.95	900	.....	No insulin
	15	—16	1.0	950	Basal +31	Insulin
	16	None	1.21	1,050	Basal +97	Insulin; best growth
23	4	—10	0.54	548	Basal +180	Insulin; best growth
24	4	± 0	0.64	650	Basal +68	Insulin
	5	None	0.73	720	Basal +79	Insulin; best growth
25	8	—25	0.75	732	Basal +98	Insulin just started
	10	None	1.00	965	Basal +34	Insulin; best growth
30	7	— 7	0.85	834	Basal +77	Insulin
	8	None	0.99	970	Basal +67	Insulin; best growth
31	9	—36	0.91	883	.....	No insulin
	11	—25	0.93	895	.....	No insulin
	11	—38	0.92	896	.....	No insulin
	12	— 4	1.15	1,115	Basal +95	Insulin
	14	None	1.26	1,210	Basal +43	Insulin; best growth

Before the use of insulin the actual basal rate averaged—20 per cent; after the use of insulin it averaged—4.5 per cent. In a few subjects the shift from a low basal rate toward the normal basal rate can be clearly seen following the use of insulin. It is unwarranted to conclude that this is due to any factor other than the ability to use more food. It is likely that the tendency of these children to gain more weight than seemed wise when first given insulin is due to the fact that their activity did not increase with their increased ability to use food.

Growth in stature and weight, on diets much below such standards as that of Holt and Fales, takes place in the presence of an increased basal metabolic rate. Since energy cannot be produced from nothing, it follows that the physical activity of these children as a group must be below the average for normal healthy children. This seems to be true except in the instances of patients 16 and 29, both of whom are active in school athletics. Their diets, however, are approximately basal, + 85 per cent, although 31 and 41 per cent, respectively, deficient in total calories. On the other hand, these two boys, aside from a short time at strenuous games, lead rather quiet lives.

DATA OTHER THAN THOSE OF GROWTH AND FOOD INTAKE

Etiology.—We have presented figures which show that in all but four of these cases there was excess weight for height, or excess stature for age, or both, at the onset of diabetes. Two of these four children were of normal proportions and suffered onset of the disease after acute infection. One patient who was light weight for height was unfortunate enough to have undergone a long course of overfeeding while at the same time having family and racial factors favoring the disease. In the fourth case, no etiologic factor can be suggested.

Factors Predisposing Toward Stature Type.—In tables 3 and 4 we have noted the stature of the parents as tall (T), medium (M), or short (S), when it could be ascertained, in order to suggest the inherited factor in regard to stature. As has been stated, irrespective of the parent's stature, nearly all these children were heavy for height, or large for age, or both, at the onset of the disease.

SUMMARY

The Bardeen average of weight for a given height roughly correlated to age has been chosen as our criterion for growth in children. Height and weight plotted on the Bardeen chart give a more valuable picture of the status of body build in the individual case. Percentage variation curves have been constructed.

Holt and Fales' standards of food requirements for normal children have been used for comparison.

The growth curves in nine cases of diabetes mellitus in children are presented as illustrative of the type of work done. An analysis of thirty-four cases of diabetes mellitus and one case of renal diabetes constitute the group analyzed.

The diets in these cases are analyzed. Data on the preinsulin course of the disease and the period of "best growth" are presented.

Data other than those of growth and food intake are discussed.

CONCLUSIONS

Of these thirty-four cases of diabetes mellitus, all but four occurred in patients who were either overweight or "overgrown," or both, at the onset of the disease. There is no other etiologic factor as significant. In two of the remaining cases, onset of diabetes occurred after acute infection. In one, no etiologic factor can be suggested.

Growth in height in children with diabetes mellitus diminishes or stops completely depending on the severity of the case, and appears to be due to lack of food.

The impulse for growth in height seems to persist longer under the adverse conditions of diabetes and to be slower in starting again than the impulse for changes in weight.

Insulin enables the patient to use sufficient food, and growthfollows.

The optimum weight for the diabetic child is the average weight for height as expressed in the Bardeen growth curve of normal children, allowing for a normal variation of $\pm$ 12 per cent.

The optimum food intake is somewhat less, possibly about two thirds of the total calories of the Holt and Fales standards.

The average proportions of carbohydrate, protein and fat are: carbohydrate, 35 per cent; protein, 15 per cent, and fat 50 per cent of the total calories. The fat and carbohydrate quotas are just about the reverse of the Holt and Fales standards. This is not necessarily the best proportion. Experience during the last two years has led us to believe that in some cases proportions nearer the normal as given by Holt and Fales yield excellent results.

Regularity in food intake and insulin dosage both as to amount and time ingestion is absolutely essential to the successful progress of the patient.

A COMPARISON OF THE EFFECTS OF SUPPLEMENTARY FEEDING OF FRUITS AND MILK ON THE GROWTH OF CHILDREN *

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A study made in this laboratory by M. S. Chaney [1] indicated that not all undernourished children are best treated by the feeding of supplementary lunches of milk and crackers, according to the widespread custom in American public schools. She found that school children given an orange daily, in place of the milk, made on the average better growth during the two eight-week periods of observation than did the milk-fed children, and both made considerably better growth than did the control group which received no supplementary lunch. The chief criterion of growth used in this study was gain in weight, although standing and sitting heights were taken also. The children examined came from homes of good economic condition, so that the total quantity and the variety of food supplied were probably adequate. Apparently also these families served plenty of milk at home, although no exact information on this point was secured.

Our first thought was that the oranges supplemented the home diet of these children by their high vitamin content, and at the same time did not detract from the appetite of the children, as the milk lunch may occasionally do. It seemed possible too that the higher alkalinity of the ash of the orange might serve a valuable purpose in offsetting the usually acid reacting mixed diet of the children.

Further work with the oranges has been carried on by Miss Chaney with Dr. K. Blunt at the University of Chicago, with rather interesting results. Chaney and Blunt [2] found that two girls, aged 10 and 11 years, respectively, when given from 600 to 700 cc. of orange juice daily in addition to a basal diet, retained considerably more calcium, phosphorus, magnesium and nitrogen than they did on the basal diet alone, and more than the added content of these elements in the orange juice could account for.

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* Received for publication, July 22, 1926.

* From the Laboratory of Household Science, the University of California.

1. Chaney, M. S.: A Comparison of the Value of Milk and Oranges as Supplementary Lunch for Underweight Children, Am. J. Dis. Child. **26**:337 (Oct.) 1923.

2. Chaney, M. S., and Blunt, K.: The Effect of Orange Juice on the Calcium, Phosphorus, Magnesium and Nitrogen Retention and Urinary Organic Acids of Gowing Children, J. Biol. Chem. **66**:829 (Dec.) 1925.

It seemed to us worth while to carry on the comparison of these supplementary feedings on a group of children known to be maintained on a constant house diet. For this reason we obtained permission to make the study in the California State Schools for the Deaf and Blind in Berkeley.

As far as possible, only children who were normal (except for their defect) were used in this study. A careful medical examination was made just previous to the beginning of the feeding, and these medical records were constantly consulted in the choice of subjects. Complete information as to the daily food served in the dining room and as to the relative appetites of the subjects was obtained through the cooperation of the nurses and attendants in charge of the children.

METHOD OF PROCEDURE

Measurements.—The following physical measurements were made on all the children studied once every four weeks, except weights, which were taken weekly: (1) weight; (2) standing height; (3) sitting height; (4) chest circumference; (5) chest expansion; (6) vital capacity, and (7) hand grip by dynamometer.

The ages of the children were recorded as that of the nearest birthday shown in the official records. Weights were taken on a Howe scale, and with shoes and coats or sweaters of the children removed. Standing height without shoes, sitting height and chest circumference were taken in the manner described by Dreyer and Hanson.[3] Chest expansion was taken on the same day as chest circumference. The child stood erect, with arms at the sides. All the air was expelled from the lungs, and the chest circumference was noted. The child was then told to take a deep breath, and the increase in chest circumference was noted. Vital capacity was measured as described by Dreyer, by means of a water spirometer, and the highest result of five trials was taken.

Measurements of hemoglobin percentage by the Tallqvist method and of alveolar carbon dioxide by the Marriott method were also made, but the results appeared untrustworthy and are not reported. Following the first alveolar carbon dioxide measurements, a group of twelve children were selected for a study of plasma bicarbonate by the method of Van Slyke and Cullen, and these results are reported in another paper.[4]

Plan of Feeding.—The extra feeding was begun, Jan. 12, 1925, and continued unchanged for fourteen weeks thereafter. At the end of this

3. Dreyer, G., and Hanson, G. F.: The Assessment of Physical Fitness, New York, Paul B. Hoeber, 1921, p. 127.

4. Morgan, A. F., and Hatfield, G. D.: The Effect of Various Supplementary Lunches on the Plasma Carbon Dioxide Capacity of Children, Am. J. Dis. Child. **32** (Nov.) 1926

TABLE 1.—*Physical Measurements of Children Given Various Supplementary*

TABLE 1.—*Physical Measurements of Children Given Various Supplementary Lunches Daily for Fourteen Weeks*

No.	Sex*	Age, Years	Weight		Height		Sitting Height		Vital Capacity		Chest Circumference		Chest Expansion		Grip Strength		Lunch	Medical Rating†	Defect
			Beginning, Lbs.	End, Lbs.	Beginning, In.	End, In.	Beginning, In.	End, In.	Beginning, Liters	End, Liters	Beginning, In.	End, In.	Beginning, In.	End, In.	Beginning, Lbs.	End, Lbs.			
1	♀	12	66.0	75.0	53.5	54.0	27.0	27.5	1.6	1.7	24.5	25	2.7	2.7	30	30	Milk	7.0	Blind
2	♀	12	67.5	70.2	52.5	53.0	27.0	27.5	1.4	1.4	25	25	1.5	3.0	40	40	Milk	8.5	Blind
3	♀	7	43.5	44.5	43.0	43.0	23.5	23.5	0.5	1.0	23	23	2.0	2.0	20	20	Milk	8.0	Blind
4	♀	8	43.0	46.2	46.5	45.5	23.0	23.0	0.9	1.3	21	22	1.2	2.5	15	20	Milk	7.7	Blind
5	♀	10	54.0	58.5	48.5	48.5	25.0	25.0	0.7	1.1	25	25.5	2.0	2.0	20	27	Milk	8.7	Blind
6	♂	11	40.0	41.5	45.5	47.0	22.5	23.5	0.5	0.7	22	22	1.5	1.5	20	20	Milk	8.7	Blind
7	♂	15	91.0	97.7	61.0	61.0	30.0	30.0	3.5	3.5	29	29	3.0	3.5	60	60	Milk	8.2	Blind
8	♂	10	55.0	59.7	48.7	49.0	25.2	26.0	1.4	1.4	23	24	2.0	2.5	20	35	Milk	7.5	Blind
9	♂	10	59.7	60.5	50.0	51.0	26.5	27.0	1.8	1.8	25	25	2.5	2.5	20	40	Milk	7.5	Blind
10	♂	14	96.7	100.0	61.0	61.0	30.5	30.5	2.7	2.7	29	29	1.7	2.5	50	75	Milk	8.0B	Blind
11	♀	7	48.5	50.5	46.0	46.0	24.0	24.0	0.7	0.8	23	23.5	1.2	2.0	20	20	Figs	7.0	Deaf
12	♀	9	58.0	64.0	49.9	49.5	26.5	26.5	1.0	1.1	24	24	1.5	1.7	20	30	Figs	7.0	Deaf
13	♀	11	68.0	71.2	53.5	54.0	29.0	29.0	1.5	1.9	26	26	2.5	3.0	40	45	Figs	8.5	Blind
14	♂	11	75.0	81.2	53.5	53.5	27.0	27.0	1.0	2.0	27.5	27.5	1.5	2.2	50	60	Figs	7.2	Blind
15	♂	7	47.0	49.0	44.5	44.5	23.0	23.0	1.0	1.0	23.5	23.5	1.5	2.0	20	20	Figs	7.5	Deaf
16	♀	11	55.0	55.0	48.0	48.0	24.0	24.0	0.9	0.9	24	24	1.7	1.5	20	20	Nothing	7.2	Deaf
17	♀	9	50.0	51.0	47.0	47.0	25.0	25.0	0.9	0.9	23	23	1.5	2.0	17	20	Nothing	7.2	Deaf
18	♀	7	47.0	48.7	46.0	46.5	24.0	24.0	0.8	0.8	23	23	1.0	1.0	30	40	Nothing	8.7	Deaf
19	♀	13	65.0	72.5	51.7	52.7	26.7	27.0	1.6	1.6	25	25	1.7	2.2	30	40	Nothing	9.0	Blind
20	♀	6	51.5	52.0	47.0	47.0	24.0	24.0	0.5	1.0	22	22	1.0	2.0	17	20	Nothing	7.7	Deaf
21	♂	11	70.0	74.5	53.5	53.5	28.0	28.0	1.5	1.6	25	25	2.5	2.5	35	45	Nothing	7.7	Deaf
22	♂	13	106.0	109.5	61.5	62.5	28.5	30.0	2.3	2.4	30.5	31	2.5	3.0	70	70	Nothing	8.7	Blind
23	♂	12	94.0	95.0	58.5	59.5	27.5	28.0	2.5	2.8	29	29	2.0	2.5	42	60	Nothing	8.5	Blind
24	♂	13	96.5	105.0	59.5	61.0	30.0	30.5	2.5	2.5	29	29.5	1.2	2.0	50	60	Nothing	8.2	Deaf
25	♀	12	54.5	58.0	50.5	50.5	28.5	25.5	0.7	0.7	23.5	23.5	1.5	1.5	17	20	Orange	9.0	Deaf
26	♀	10	56.5	59.0	52.0	52.0	27.0	27.0	1.8	1.8	25	25	2.5	3.0	25	30	Orange	7.0B	Deaf
27	♀	7	25.5	29.0	39.0	39.5	21.5	22.0	0.1	0.5	18	19	1.0	1.2	10	20	Orange	7.7	Deaf
28	♀	10	62.5	63.5	49.5	50.0	25.5	25.5	1.3	1.3	25	25	2.5	2.5	20	25	Orange	7.5B	Deaf
29	♀	10	55.5	56.5	49.5	49.5	25.5	25.5	1.3	1.3	23	24	1.7	2.2	20	30	Orange	8.0	Deaf
30	♀	8	50.2	53.7	47.5	48.0	24.0	24.5	1.0	1.0	23	23	1.5	1.5	17	20	Orange	8.7	Deaf
31	♀	9	55.0	56.0	48.0	48.5	25.0	25.0	1.0	1.2	23	23	2.0	2.0	20	25	Orange	7.5	Deaf
32	♀	11	62.5	65.0	52.5	53.0	26.5	26.5	1.3	1.4	24	24	1.2	3.5	35	35	Orange	...	Deaf
33	♂	7	48.5	51.5	44.5	44.5	23.0	23.0	0.8	0.8	23	23	1.0	1.5	20	25	Orange	8.2B	Deaf
34	♂	10	79.0	81.0	57.0	57.0	28.0	28.0	1.2	1.4	25	25.5	1.5	2.0	50	55	Orange	8.5	Deaf
35	♂	14	85.2	93.7	59.0	60.0	28.5	29.0	2.7	2.7	27	27.5	2.7	3.5	50	55	Orange	7.5	Deaf
36	♂	12	57.5	60.0	51.0	51.0	26.5	26.5	1.1	1.3	24	24	1.5	1.5	25	25	Orange	7.7	Deaf
37	♂	7	56.5	58.0	47.5	48.0	23.5	24.0	1.0	1.0	24	24	1.5	1.5	17	25	Orange	8.5	Deaf
38	♂	10	63.5	66.0	53.0	53.5	27.0	27.5	1.5	1.8	25	25	2.0	2.5	35	40	Figs	8.1B	Blind
39	♀	6	43.5	43.0	45.0	45.0	24.0	24.0	0.9	0.9	21	21.5	1.5	1.5	15	20	Figs	7.6	Deaf
40	♀	14	65.7	67.0	54.5	55.5	28.5	29.0	2.1	2.2	23.5	25.5	2.5	2.5	20	25	Figs	9.0	Blind
41	♀	9	34.0	35.5	41.5	43.2	20.5	21.5	0.6	0.8	22	22	2.5	2.5	5	10	Milk	7.3B	Blind
42	♂	6	39.2	41.0	43.0	43.0	22.0	22.0	0.8	0.8	22	22.5	1.2	1.5	15	20	Figs	8.0B	Deaf
43	♂	13	67.5	69.5	49.5	51.0	25.5	27.0	1.8	1.8	27	27	2.5	2.5	40	40	Milk	8.5	Blind
44	♂	12	85.5	85.0	59.0	59.0	30.0	30.5	1.9	2.0	24	24	2.5	2.5	60	65	Figs	7.3	Deaf
45	♂	9	75.0	78.5	54.5	55.0	26.5	27.0	1.8	2.0	27	27	2.7	2.7	40	45	Milk	9.0	Blind
46	♂	10	81.0	82.5	51.5	52.5	27.0	27.0	1.5	2.0	27	27	1.5	2.0	45	45	Nothing	8.3	Blind
47	♂	10	84.0	87.5	51.5	51.5	27.0	27.0	0.8	0.9	30	31	1.5	1.5	30	30	Nothing	8.6B	Blind

* In this column, ♀ indicates female; ♂, male.

† Medical Rating: 9, very good; 8, good; 7, fair; 6, poor; 5, very poor. Organic defect present, B. Condition of teeth, nose, throat, glands and posture were rated separately and averaged.

period, the type of supplementary lunch was changed in some cases, and the observations were continued for four weeks longer. In table 1 are tabulated all the physical measurements of the forty-seven children, taken at the beginning and end of the fourteen weeks' period.

The supplementary lunches were served at the midmorning recess. The milk lunch consisted of one-half pint of grade A pasteurized milk daily; the orange lunch of one medium large, sweet navel orange; the fig lunch of four large pulled figs of the best grade obtainable. The average caloric value of the milk lunch was 160, of the orange lunch 100, and of the fig lunch 200.

Method of Grouping.—All the children in the schools who were underweight, according to the Baldwin-Wood standard, were chosen as subjects of the experiment, and, in addition, about an equal number of normal or overweight children, because the number of those underweight was rather small. As may be seen in table 3, a larger number of underweight children were placed in the orange group than in any of the others. Unfortunately, there were no underweight subjects assigned to the control group. Thus the odds in favor of good growth on these various lunches might be assumed to be in this order: Control, milk, fig and orange. These differences are indicated by the average medical ratings shown in table 3. The choice between the blind and deaf children was made regardless of the nature of their defects, and, consequently, as may be noted also in table 3, a somewhat uneven distribution resulted.

Seven of the children, numbers 25, 26, 27, 4, 6, 40 and 41, were underweight even when allowance was made for the amount of variability calculated by Faber [5] as normal to their sex and size. Four others, numbers 14, 43, 46 and 47, were overweight by the same criterion. All of the others fell within the limits of the variability for normal children described by Faber.

Effect of the Feeding on Weight.—In table 2 a comparison is made of the state of nutrition of the children at the beginning and end of this period, as judged by three standards, namely, the Baldwin-Wood tables,[6] the pelidisi of Pirquet,[7] and the Dreyer weight tables.[8] No satisfactory general agreement is observable among the figures obtained by application of these three standards, and striking differences are seen in certain cases of unusually built children. For instance, number 47, because of large chest measurement, appears actually 4 per cent under-

5. Faber, H. K.: Variability in Weight for Height in Children of School Age, Am. J. Dis. Child. **30**:328 (Sept.) 1925.

6. Baldwin, B. T., and Wood, T. D.: Mother and Child, 1923, July Suppl. Am. Child Health Assoc., Washington, D. C.

7. Pirquet, C.: An Outline of the Pirquet System of Nutrition, London, Baillière, Tindall & Cox, 1922, p. 96.

TABLE 2.—*Growth of Children Given Various Supplementary Lunches for Fourteen Weeks*

No. of Child	Percentage Over or Under Normal Weight Standard of Baldwin and Wood			Pelidisi			Percentage Over or Under Dreyer Standard			Percentage of Expected Gain (Holt Standard)	Total Weight Gain, Lbs.
	Beginning	End	Change	Beginning	End	Change	Beginning	End	Change		
1	− 6.6	+ 5.6	+12.2	98	100	+2	+ 1.7	+ 8.2	+ 6.5	300	9.0
2	± 0	+ 1.8	+ 1.8	98	97	−1	± 0	+ 0.1	+ 0.1	90	2.7
3	+ 6.1	+ 8.5	+ 2.4	98	98	±0	− 7.4	− 5.3	+ 2.1	99	1.0
4	−12.2	− 5.6	+ 6.6	99	101	+2	+10.2	+12.1	+ 1.9	290	3.2
5	+ 0.9	+ 7.3	+ 6.4	99	102	+3	−10.9	− 7.4	+ 3.5	300	4.5
6	−14.9	−17.0	− 2.1	99	97	−2	+ 2.3	− 0.8	− 3.1	100	1.5
7	− 9.0	− 2.2	+ 6.8	98	100	+2	+ 1.6	+ 9.1	+ 7.5	223	6.7
8	± 0	+ 8.6	+ 8.6	97	98	+1	+12.2	+ 9.5	− 2.7	313	4.7
9	+ 3.0	− 0.8	− 3.8	96	95	−1	+ 0.6	− 1.2	− 1.8	53	0.8
10	+ 1.0	+ 1.0	± 0	98	99	+1	+ 6.8	+10.3	+ 3.5	110	3.3
11	+ 3.2	+ 7.4	+ 4.2	99	100	+1	+ 0.2	± 0	− 0.2	181	2.0
12	+ 5.1	+13.2	+ 8.1	95	99	+4	− 4.1	+ 5.7	+ 9.6	271	6.0
13	− 2.1	+ 0.3	+ 2.4	92	93	+1	−15.7	−11.7	+ 4.0	145	3.2
14	+10.9	+18.6	+ 7.7	103	105	+2	+ 6.5	+15.3	+ 8.8	413	6.2
15	+ 4.2	+ 8.9	+ 4.7	102	104	+2	+ 4.9	+ 9.3	+ 4.4	181	2.0
16	+ 3.7	+ 3.7	± 0	103	103	±0	+ 4.5	+ 4.5	± 0	0	0
17	± 0	+ 2.0	+ 2.0	96	97	+1	− 2.5	− 0.6	+ 1.9	66	1.0
18	± 0	+ 0.5	+ 0.5	97	99	+2	− 2.8	+ 0.6	+ 3.4	155	1.7
19	+ 3.0	+ 2.0	− 1.0	97	99	+2	− 2.3	+ 7.5	+ 9.8	341	7.5
20	+ 3.0	+ 4.0	+ 1.0	101	101	±0	+16.0	+17.3	+ 1.3	136	0.5
21	+ 2.1	+ 8.7	+ 6.6	96	97	+1	+ 8.0	+14.2	+ 6.2	300	4.5
22	+ 6.5	+ 4.7	− 1.8	108	104	−4	+18.9	+11.8	− 7.1	116	3.5
23	+ 8.0	+ 4.9	− 3.1	107	106	−1	+20.0	+18.3	− 1.7	66	1.0
24	+ 6.0	+ 8.2	+ 2.2	100	101	+1	+ 9.0	+13.0	+ 4.0	283	8.5
25	−14.1	− 8.6	+ 5.5	97	99	+2	+ 3.8	+10.4	+ 6.6	151	3.5
26	−11.7	− 7.8	+ 3.9	93	94	+1	−16.1	−12.4	− 3.7	166	2.5
27	−25.0	−19.4	+ 5.6	90	90	±0	+ 0.4	+ 0.3	− 0.1	318	3.5
28	+ 8.9	+ 7.6	− 1.3	102	104	+2	+ 0.6	+ 2.2	+ 1.6	66	1.0
29	− 3.4	− 1.7	+ 1.7	98	99	+1	+ 4.9	− 0.1	− 5.0	66	1.0
30	− 1.4	+ 3.3	+ 4.7	100	100	±0	+ 4.3	+ 8.0	+ 3.7	318	3.5
31	+ 7.8	+ 4.6	− 3.2	100	101	+1	+ 7.2	+ 9.2	+ 2.0	66	1.0
32	− 6.0	− 4.4	+ 1.6	98	99	+1	+ 3.3	+ 7.4	+ 4.1	166	2.5
33	+ 7.7	+14.4	+ 6.7	104	106	+2	+12.2	+19.2	+ 7.0	272	3.0
34	+ 1.2	+ 5.2	+ 4.0	99	101	+2	+21.9	+21.8	− 0.1	133	2.0
35	− 5.2	− 0.2	+ 5.0	101	103	+2	+15.3	+25.0	+ 9.7	283	8.5
36	− 5.7	− 1.6	+ 4.1	95	97	+2	+ 2.6	+ 7.1	+ 4.5	166	2.5
37	+ 9.7	+ 9.4	− 0.3	107	104	−3	+19.7	+19.9	+ 0.2	136	1.5
38	− 5.2	− 3.6	+ 1.6	97	96	−1	+ 3.6	+ 4.7	+ 1.1	200	2.5
39	− 3.3	− 4.4	− 1.1	96	96	±0	+ 5.9	+ 0.4	− 5.5	0	0.5
40	−14.7	−16.7	− 2.0	101	100	−1	− 0.9	−14.2	−13.3	188	0.3
41	−10.5	−15.4	− 4.9	103	97	−6	− 5.8	− 7.0	− 1.2	114	1.5
42	− 4.4	± 0	+ 4.4	101	102	+1	+ 3.1	+ 4.0	+ 0.9	180	1.8
43	+16.8	+13.9	− 2.4	104	100	−4	+ 6.7	+ 1.4	− 5.3	80	2.0
44	− 3.9	− 6.0	− 2.1	96	92	−4	+25.7	+17.9	− 7.8	0	0.5
45	+ 5.6	+ 9.0	+ 3.4	105	104	−1	+12.2	+14.6	+ 2.4	233	3.5
46	+29.6	+25.9	− 4.3	105	105	±0	+18.2	+20.4	+ 2.2	120	1.5
47	+34.4	+40.0	+ 5.6	107	110	−3	+ 3.2	+ 1.6	− 1.6	280	3.5

TABLE 3.—*Average Changes by Three Standards in Percentage Under and Overweight of Children Given Various Supplementary Lunches*

Lunch	No. in Group	Underweight at Beginning by Baldwin-Wood Scale		Average Change in Weight for Height and Age in 14 Weeks				Average Gain in Pounds in 14 Wk.	Average per Cent Gain Above That Expected	Average of Medical Ratings	Percentage of Groups Having Organic Defects	Deaf, per Cent	Blind, per Cent
		No.	%	By Baldwin-Wood Standard	By Pirquet Standard (Pelidisi)	By Dreyer Standard	Average of the Three Changes						
Milk	13	5	38	+2.7	−0.8	+1.0	+1.1	3.4	77	8.0	15.4	0	100
Fig	10	5	50	+2.8	+0.5	+0.2	+1.2	2.3	67	7.7	20.0	60	40
Orange	13	8	62	+2.9	+1.0	+2.3	+2.0	2.7	77	7.9	25.0	100	0
Control	11	0	0	+0.7	+0.4	+1.6	+0.9	3.0	69	8.1	9.0	54	46

weight by the Dreyer standard, although 40 per cent overweight by the Baldwin-Wood table. Number 35, although slightly underweight by the Baldwin-Wood table, is 25 per cent overweight by the Dreyer standard, and has a pelidisi of 103. This is due to an unusually short stem length as compared with standing height and weight. About one-half of the children showed large differences between Dreyer's normal weights as calculated from chest circumference and stem length, respectively. These discrepancies appear quite as often in boys as in girls, so that breast development in the latter can scarcely account for them.

The change in percentage over or under the three weight standards brought about during the fourteen weeks of observation is also recorded in table 2, and is summarized by lunch groups in table 3. The orange fed group made apparently the largest average gains as judged by these three standards, the milk and fig groups came next, and the control

TABLE 4.—*Summary of Gains in Physical Measurements of Children Given Various Supplementary Lunches Daily for Fourteen Weeks*

		Proportion of Groups Making Gains in														
		Weight		Height		Sitting Height		Vital Capacity		Chest Circum.		Chest Expansion		Grip Strength		Average of All Gains,
Lunch	No. in Group	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	%
Milk.....	13	13	100	8	61	8	61	6	46	4	30	5	38	7	53	55.5
Fig.....	10	8	80	5	50	3	30	7	70	5	50	7	70	7	70	60.0
Orange.....	13	13	100	7	54	4	30	5	38	4	30	7	54	11	84	55.7
Control....	11	9	81	6	55	4	36	5	45	3	27	7	63	7	63	52.8

group last. The same general order holds for average percentage gain above that expected, except that the fig group is lower here than the controls. In total average gain in pounds the order is milk, control, orange and fig.

Effect of Illness.—Five children of the fig group, numbers 38, 39, 40, 42 and 44, were victims of a mild attack of influenza and were in the hospital from one to five weeks during the experiment. During this time they received no figs, and naturally suffered considerable setbacks in weight. If these five were eliminated from the averages, the other five children remaining in the fig group would be found to show much higher weight gains than any of the other three groups, 198 per cent above the expected gain and 3.8 pounds per child total gain for the fourteen weeks. Only one of the children in the milk group, number 41, two in the orange group, numbers 28 and 29, and none in the control group were in the hospital for illness during the experiment.

Effect of the Feeding on Measurements Other Than Weight.—In table 4 are shown the number of children in each of the groups who made gains in the various measurements taken.

Very little difference is observable among the groups in gains in standing height. Apparently at these ages from 50 to 60 per cent of all children grow at least one-half inch in fourteen weeks.

In sitting height there is more variation, and here the milk-fed group distinctly leads the others. The variation in accuracy of measurement may be greater in this than in standing height. It must be remembered, however, that all these measurements were made at least four times on all the children.

In vital capacity, the fig group leads the others. Practically all the children in every group gave figures for vital capacity considerably below the standards set by Dreyer. Whether this is due to a consistent difference in record by the instrument used, or to an actual deficiency in the children, it is impossible to judge. We are inclined to the former view, however, in view of the relatively normal figures shown by the subjects in all other respects. In chest circumference the fig group again leads, as it does in chest expansion. These two measurements, along with vital capacity, all seem physiologically related, and may represent a real advantage presented by the fig feeding.

In muscle strength as indicated by the dynamometer grip, there is very little difference in number of increases shown by the various groups, the orange being in the lead.

It is evident that variations in the composition of groups used for such a comparative feeding test as this affect the results notably. Even under the handicap of such unevenness of amount of undernutrition as is exhibited by these four groups, however, certain advantages of supplementary feeding of fruits and milk are evident. The greater growth in stem length of the milk-fed group, and in all chest measurements of the fig-fed group, seems to indicate real differences from the other groups. Change in weight for height, age, stem length and chest circumference as measured by the three standards, Baldwin-Wood, Pirquet and Dreyer, seem to show a real advantage in the orange feeding similar to that shown previously by Chaney.[1]

THE COMPOSITION OF THE HOUSE DIET

The meals served the children in the institution were observed, and an analysis of their make-up throughout the eighteen weeks of the experiment is given in table 6. There can be no criticism of the total caloric content of the house diet, for an abundance was served at every meal. The diet is rather overbalanced as to carbohydrate, especially in cereal and starchy foods. Bread, cake, pastry and puddings were served with excessive frequency. Many combinations, especially those for the evening meal, seem rather difficult of digestion, even for adults. Oyster

stew, potato salad and bologna sausage, or cream of tomato soup, frankfurters or sardines, lyonnaise potatoes and fruit gelatin were not unusual offerings.

The total amount of milk offered was certainly sufficient to meet any standard. Indeed, a quart of milk per day per child was conscientiously

TABLE 5.—*Rate of Gain or Loss in Weight of Children Changed from Milk to Orange Lunches and Vice Versa*

Supplementary Lunch	No. of Child	Weight in Pounds			Weekly Rate of Gain	
		Beginning	End of 14 Weeks	End of 4 Weeks	14 Weeks Milk, Lbs.	4 Weeks Orange, Lbs.
Milk for first 14 weeks, orange for 4 weeks	7	91.0	98.0	99.5	0.50	0.37
	10	95.7	98.0	100.5	0.16	0.62
	43	67.5	69.5	69.0	0.14	—0.12
	9	59.7	59.5	61.0	—0.01	0.37
	6	40.0	40.7	40.0	0.05	—0.17
	41	34.0	35.2	36.0	0.08	—0.20
	3	43.5	44.0	43.0	0.03	—0.25
	Average				0.13	0.14
					Orange	Milk
Orange for first 14 weeks, milk for 4 weeks	36	57.5	58.5	60.0	0.07	0.37
	33	48.5	51.0	49.5	0.18	—0.37
	35	85.2	95.0	95.0	0.70	0.00
	26	56.5	58.0	58.2	0.10	—0.05
	25	54.5	58.5	57.7	0.28	—0.20
	27	28.0	28.2	28.5	0.01	0.07
	31	55.5	55.5	57.2	0.00	0.42
	Average				0.16	0.05

TABLE 6.—*Regular Meals Served Subjects During Experimental Period*

Food	Average Times per Week	Totals per Week	
Fruit:			
Oranges	1.5		
Other fresh fruit	2.1		
Canned or cooked fruit	4.5	Fruit	8.1
Vegetables:			
Fresh	8.3	Raw fruit and vegetables	6.4
Potatoes	8.1		
Canned vegetables	4.4		
Tomatoes, usually canned	2.8		
Dried legumes	2.0	Vegetables	25.6
Cereal Foods:			
Pastry, cake, etc.	5.7		
Cereals	7.0		
Bread	21.0	Cereal foods	33.7
Animal Foods:			
Butter	21.0		
Milk	21.1		
Ice cream	2.0		
Cocoa (milk)	9.8	Milk foods	32.9
Eggs	3.7		
Cheese	1.4		
Meat	9.0		
Fish	1.7		
Gelatin	2.0	Animal protein foods	17.8

included in the diet. It is doubtful whether the children partook of the milk in so uniform a manner, however. No systematic attempt was made to see that each child ate any given amount of any food, but merely had set before him plenty of the kind of foods indicated.

The amount of fresh and canned vegetables served seems adequate, but the variety and amount of fruit given is rather surprisingly small. In particular, the antiscorbutic fresh raw fruits and vegetables were

included less often than is usually considered desirable. Enough animal protein appears on these lists to meet the usual requirement. On the whole, the diet was well above the average for institutions, and certainly no criticism could be made of those who planned and purchased the food. The chief difficulty seemed to lie in too great a desire to meet the demands of the children for the less digestible foods.

The Ash Reaction of the Diet.—The ash reaction of the total diet was calculated for a number of days, including amounts of each of the foods found to represent the average appetite of the children. An example is given in table 7. The balance in each case was acid, usually showing an excess of about 40 cc. normal acid. This is much greater than the 12 to 18 cc. excess recorded by Sherman [8] as characteristic of American dietaries. According to Shohl,[9] who recalculated the results

TABLE 7.—*Estimate of Typical Day's Dietary as to Total Fuel Value and Reaction*

Meal	Food	Fuel Value Calories		Equiv. in Cc. Normal Acid	Equiv. in Cc. Normal Base	
Breakfast	Apple sauce	100		.....	5.98	
	Oatmeal	200		4.84	.....	
	Toast	150		4.05	.....	
	Milk	150		.....	5.16	
			600			
Dinner	Veal	200		17.55	.....	
	Rice	150		4.95	.....	
	Carrots	50		.....	11.95	
	Peaches	125		.....	6.01	
	Bread and butter	150		4.05	.....	
	Milk	150		.....	5.16	
			825			
Supper	Corn	200		3.54	.....	
	Eggs	100		7.55	.....	
	Lima beans	200		24.16	.....	
	Chocolate bread pudding	200		5.40	.....	
	Bread and butter	200		4.05	.....	
	Milk	150		.....	5.16	
			1,050			
Total			2,475	80.14	39.42	
Excess acid, equivalent in cc. normal solution						40.76
Supplementary lunches	Milk	160		.....	4.7	
	Orange	100		.....	10.9	
	Figs	200		.....	64.6	

of Sherman [8] and of Sawyer, Baumann and Stevens,[10] growing children retain from 10 to 15 cc. per kilogram of body weight per day of 0.1 normal base in excess of acid. The diets given these children then are excessively acid in reaction, and there can be little surprise accordingly that the plasma bicarbonate figures found for a number of these children, and given in another publication,[4] are so low. The very high basicity reported for dried figs [11] and the relatively high basicity of oranges as compared with that of milk, would offer a tempting explanation of

8. Sherman, H. C.: Chemistry of Food and Nutrition, New York, The Macmillan Company, 1926, p. 271.

9. Shohl, A. T.: Mineral Metabolism in Relation to Acid-Base Equilibrium, Physiol. Rev. **3**:514, 1923.

10. Sawyer, M.; Baumann, L., and Stevens, F.: Studies of Acid Production: II. The Mineral Loss During Acidosis, J. Biol. Chem. **33**:103, 1918.

11. Sherman, H. C.: Food Products, New York, The Macmillan Company, 1914, p. 567.

the gains made by the orange and the fig groups. The increases in chest measurements in the fig group might possibly result from increased carbon dioxide capacity of the alveolar air, accompanied by deeper and more regular respiration.

Results of Exchange of Orange and Milk Groups.—In order to test the orange and milk feeding on the same children, at the end of the fourteen weeks' period seven children were changed from the orange to the milk group, and seven were changed from the milk to the orange group. The results on weight increases are shown in table 5. The children who averaged a gain of 0.13 pound per week for fourteen weeks on milk gained 0.14 pound per week for four weeks on orange. The children who gained 0.16 pound per week on orange gained only 0.05 pound per week on milk. The advantage in the latter case is plainly in favor of the orange, while in the former case the difference is probably negligible.

Changes in the other measurements besides weight were also noted, but because of the short duration of the second period, four weeks, we felt that little significance could be attached to them. However, four out of the seven children changed from milk to orange made gains in standing height and chest circumference, and two made gains in sitting height. Only two out of the seven children changed from orange to milk made gains in standing height. None made gains in sitting height, and four made gains in chest circumference. The other changes in measurements were so scattering as to be negligible.

SUMMARY

1. Forty-seven children living in the California State Schools for the Deaf and Blind were repeatedly examined for physical indexes of growth during a total of eighteen weeks in the spring of 1925. During this period, thirteen of these children were given one-half pint of milk each per day as supplementary lunch, thirteen were given one medium large orange each, ten were given four pulled figs each, and eleven were used as controls.

The degree of underweight and lack of fitness of these groups when the feeding was begun were in decreasing order, orange, fig, milk and control. Five of the fig group, two of the orange, one of the milk, and none of the control group suffered an attack of influenza during the experiment. All of the children were served the same regular meals throughout in the institution dining room.

2. Judged by the Baldwin-Wood standard of nutrition following fourteen weeks of supplementary feeding, the milk, orange and fig groups had made almost exactly the same amount of improvement. The control group had increased only about one-fourth as much as the other three. The Baldwin-Wood standard involves standing height, weight and age.

Judged by the Pirquet (pelidisi) standard, the orange group made the most improvement, the fig and control groups about one-half as much, and the milk group had fallen off a little. The Pirquet standard involves sitting height as well as body weight. It should be noted that the milk group made the most gains in sitting height, thus in part explaining the lowering of pelidisis.

Judged by the Dreyer standard, the orange group again made the most growth, the control and milk each about one-half as much, and the fig group the least. The Dreyer standard involves chest circumference and sitting height as bases for normal weight. The greater gains in chest circumference made by the fig group may in part account for their apparently low gains in weight.

An average of all these gains shows the orange group first, the fig and milk next and the control group last.

The gross average gain in pounds was largest for the milk group, and the average percentage gain in weight above that expected according to the Holt standard was largest for the orange and milk groups.

3. Seven children were changed from the milk to the orange lunch and seven from the orange to the milk lunch at the end of fourteen weeks, and the feeding continued for four weeks longer. The rate of gain in weight was considerably decreased in those changed from orange to milk, but very little affected in those changed from milk to orange.

4. The percentage of the original groups showing increases after fourteen weeks in standing and sitting heights was greatest in the milk group; in chest circumference, chest expansion and vital capacity in the fig group, and in hand grip strength in the orange group.

Of these six measurements, real differences in proportion of increase are thought to be shown only in sitting height in which the milk group excelled, and in chest circumference, chest expansion and vital capacity in all of which the fig group excelled. The average of all measurements showed the number of increases in the fig group to be the largest.

5. The composition of the house diet is shown to be excellent as to milk and animal protein content, fair as to vegetable content, but somewhat deficient in fruits, especially of the fresh raw variety. The ash reaction of the diet as eaten was undoubtedly acid, and it is possibly in the supplementing of this as well as of the antiscorbutic content of the diet that the oranges were effective. The value of figs may lie in their high basic ash and possibly also in their laxative action. The value of extra milk for these children must lie chiefly in the extra caloric value, as well as the supplementing of a possibly uneven milk intake at meals.

6. A possible correlation is suggested between the increases in chest measurements of the fig group and increased carbon dioxide capacity of the plasma resulting from the excess basicity of the fig ash.

CUTANEOUS ADMINISTRATION OF COD LIVER OIL *

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The value of cod liver oil in preventing and in curing rickets has been heralded with extreme enthusiasm during the past few years, and recent literature has been filled with reports of investigations concerning its action, and concerning its apparently close relationship to the action of ultraviolet irradiation. As might be expected, the interchangeability of cod liver oil and ultraviolet therapy has been overrated, for rational investigations, notably those of Wilson,[1] have shown that infants receiving apparently sufficient doses of cod liver oil will develop rickets in almost as high a percentage of cases as will those not so treated. The value of the oil seems to be one of control rather than of prevention, the infants receiving it manifesting rickets that healed earlier than that in the infants of the control series.

Despite the fact that the serum concentration of calcium, and especially of inorganic phosphorus, has generally been accepted as a criterion of the presence of active rickets, Wilson found these values to be normal, even with early clinical and roentgenographic evidence of the disease.

The oral administration of cod liver oil seems to have been the method almost exclusively employed. Soames,[2] however, injected it intraperitoneally into rats on a diet producing rickets and into others on a diet deficient in the fat soluble organic factor, and found that partial protection was afforded against the diet producing rickets, and that improved growth and calcification were obtained in those rats on the diet deficient in the fat soluble vitamin. He concluded that the site of action of cod liver oil is not limited to the intestinal tract. This is contrary to the views of Zucker and Matzner,[3] who found that the subcutaneous injection of the active principle of cod liver oil produced no healing of rickets and no change in the reaction of the stool.

Since the importance of the skin as an independent organ of assimilation is becoming better recognized and has long been appreciated in therapeusis, it seemed logical to determine, if possible, whether or not the results commonly attributed to cod liver oil could be obtained by its

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* Received for publication, Sept. 10, 1926.

* From the Children's Medical Department of the Massachusetts General Hospital, and the Department of Pediatrics of the Harvard Medical School.

1. Wilson, May G.: The Prevention of Rickets, Am. J. Dis. Child. **31**:603 (May) 1926.

2. Soames, K. M.: Biochem. J. **18**:1349, 1924.

3. Zucker, T. F., and Matzner, M. J.: Chem. Abstr. **19**:1586, 1925.

cutaneous administration; namely, the raising of a lowered blood phosphorus concentration to its normal value.

Infants in the wards of the Children's Medical Service were selected who had been untreated for rickets, and who showed inorganic blood phosphorus concentrations below the normal level, regardless of whether

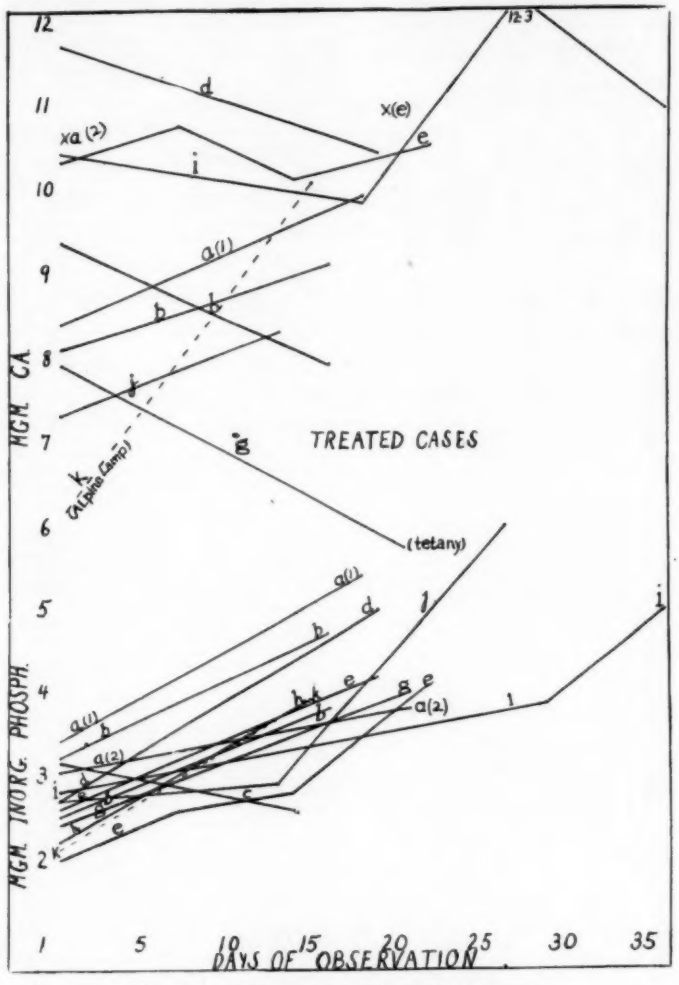

Chart 1.—Observations in treated cases

or not clinical signs of rickets were present. The majority, however, showed obvious signs of rickets. Gauze pads, saturated with 1 tablespoonful of oil, were applied to the abdomens of these infants and were held in place with flannel binders. These pads were renewed daily during the course of the experiments. The results in treated and in control cases are shown on the accompanying charts.

No attempt was made to demonstrate the healing of rickets. The experiments had to do solely with the calcium and phosphorus plasma values, analyses for which were performed by Miss Margaret E. Moriarty, using the Kramer-Tisdall method for calcium and the Briggs modification of the Bell-Doisey method for inorganic phosphorus.

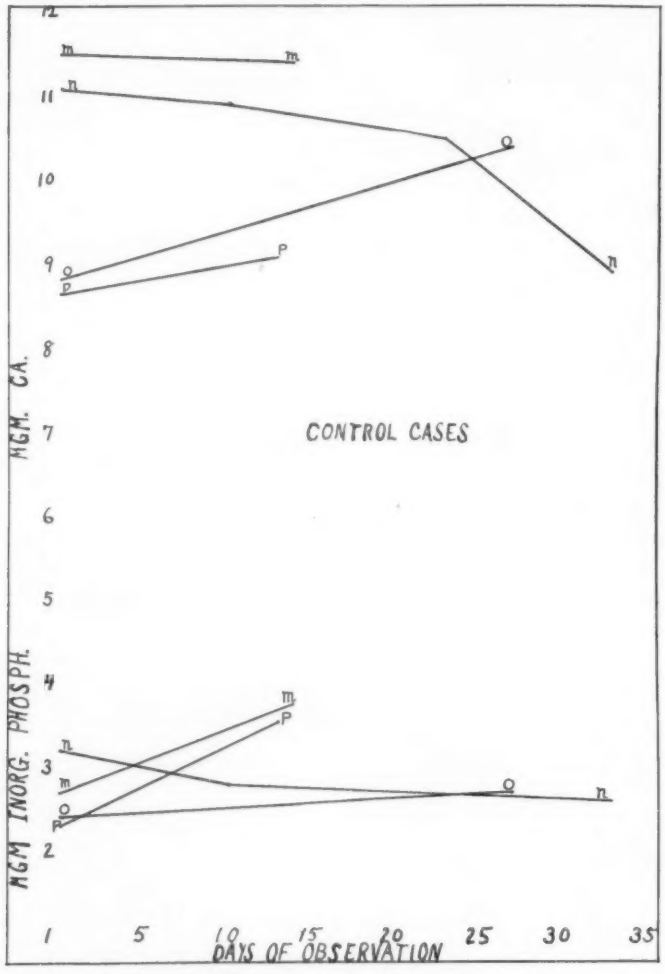

Chart 2.—Observations in control cases.

RESULTS

It will be noted that of eleven patients treated (one of whom was treated on each of two admissions to the hospital), all but one showed a marked rise in the inorganic phosphorus in from two to three weeks of treatment. The patient showing a fall in the phosphorus concentration unfortunately could not be observed longer than fourteen days.

This condition was complicated by scurvy. The fewer calcium determinations are of less significance, although of eight cases observed, only two showed an actual decline to below the normal level. Of four untreated control cases, two showed a rise in the phosphorus to approximately the normal value within two weeks, and two, observed for twenty-seven and thirty-five days, respectively, showed no improvement in this respect.

COMMENT

Although no conclusions should be drawn from this small series of cases, various possibilities suggest themselves.

The influence of cod liver oil on blood phosphorus concentration is still a matter of investigation and has not yet been thoroughly cleared up. If this influence is actual, it would seem that cod liver oil absorbed through the skin is capable of exerting it.

Two control cases suggest the possibility that blood phosphorus tends to approach the normal, even when antirachitic measures are rigidly withheld.

The relationship of these factors to clinical rickets presents still further complications.

THE PRODUCTION OF A LOWERED CARBOHYDRATE TOLERANCE IN DOGS *

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In a previous communication,[1] it was shown that glucose injected into the blood stream of infants with diarrhea, infection or acute intestinal intoxication was not removed with the normal rapidity, and that this derangement of the carbohydrate metabolism was not primarily associated with a deficient production of insulin. In the present study, an attempt was made to determine what factors were responsible for this derangement.

The animals used were puppies. In the preliminary experiments it had been found that conditions which produced no change in adult animals produced profound changes in young animals. As the object of the experiment was to produce conditions similar to those encountered in infants if possible, the importance of the age of the animals used is at once apparent. The blood sugar concentration in normal puppies was determined after the intravenous administration of 10 cc. per pound of body weight of a 10 per cent solution of glucose. The results obtained are given in table 1. The procedure used in the determination of the blood sugar concentration is based on the Shaffer-Hartmann method, and has been described elsewhere.[2] In no instance was food given to the puppies during the eighteen hours previous to the glucose tolerance tests. The effect on the glucose tolerance values produced by the previous subcutaneous administration of a bacterial toxin (diphtheria toxin) and a chemical toxin (histamine) is given in table 2. The effect produced by dehydration is given in table 3; while in table 4 are the results obtained in an attempt to produce diarrhea by the oral administration of magnesium sulphate, powdered jalap and castor oil. The effect of an elevation of temperature without any evidences of a toxemia on the glucose tolerance values is given in table 5. The summary of the averages of the values recorded in tables 1 to 4 are given in table 6. For comparison, the average values obtained with infants [1] are included in this table.

* Received for publication, Aug. 6, 1926.

* From the Nutritional Research Laboratories of the Hospital for Sick Children and the Sub-Department of Pediatrics, University of Toronto.

1. Tisdall, F. F.; Drake, T. G. H., and Brown, A.: Carbohydrate Metabolism of Infants with Diarrhea, Am. J. Dis. Child. **30**:837 (Dec.) 1925.

2. Tisdall, F. F.; Drake, T. G. H., and Brown, Alan: Carbohydrate Metabolism of Infants, J. Lab. & Clin. Med. **10**:704 (June) 1925.

The Effect of a Bacterial Toxin.—The results recorded in tables 2 and 6 illustrate quite clearly that a bacterial toxin such as diphtheria toxin may impair definitely the ability of the organism to remove injected glucose from the blood stream at a normal rate. The puppies were

TABLE 1.—*The Blood Sugar Concentration in Normal Puppies Before and After the Administration of 10 cc. per pound of Body Weight of a 10 Per Cent Solution of Glucose*

No. of Dog	Weight of Dog, Pounds	Blood Sugar Before Administration of Glucose, per Cent	Blood Sugar ½ Hour After, per Cent	Blood Sugar 1 Hour After, per Cent	Blood Sugar 1½ Hours After, per Cent
1	14	0.087	0.173	0.105	0.097
3	8	0.109	0.142	.....	0.115
4	12	0.109	0.135	0.102	0.098
5	15	0.105	0.112	0.098	0.106
6	8	0.085	0.155	0.088	0.090
7	11	0.100	0.095	0.100	0.100
53	7	0.073	0.173	0.090	0.058
54	10	0.095	0.185	0.083	0.112
55	11	0.093	0.188	0.122	0.083
56	11	0.070	0.194	0.100	0.065

TABLE 2.—*The Effect of the Previous Subcutaneous Administration of a Bacterial Toxin (Diphtheria Toxin) and a Chemical Toxin (Histamine) to Puppies on the Blood Sugar Values Found Before and After Administration of 10 cc. of a 10 Per Cent Solution of Glucose Per Pound of Body Weight*

Puppy No.	Puppy Weight	Substance of Treatment Given	Blood Sugar Before Administration of Glucose, per Cent	Blood Sugar ½ Hour After, per Cent	Blood Sugar 1 Hour After, per Cent	Blood Sugar 1½ Hrs. After, per Cent	Non-protein Nitrogen in Serum, Mg. per 100 Cc.	Condition of Dog
1	14	30 minimal lethal doses diphtheria toxin 24 hours previous to glucose	0.115	0.353	0.233	0.194	30.7	Toxic
6	8	30 minimal lethal doses diphtheria toxin 30 hours previous to glucose	0.095	0.368	0.220	0.141	60.0	Toxic
19	12	7.5 minimal lethal doses diphtheria toxin 72 hours and 24 hours previous to glucose	0.115	0.332	0.206	0.151	120.0	Toxic
17	12	210 mg. histamine divided into 4 doses during the 24 hours previous to glucose	0.112	0.323	0.225	0.129	20.0	Active, almost normal
20	18	405 mg. histamine divided into 4 doses during the 30 hours previous to glucose	0.110	0.277	0.148	0.125	26.0	Apparently normal
24	16	150 mg. histamine divided into 5 doses during the 30 hours previous to glucose	0.100	0.353	0.211	0.129	18.1	Apparently normal
26	17	245 mg. histamine divided into 4 doses during the 12 hours previous to glucose	0.116	0.383	0.298	0.220	42.0	Toxic
27	20	320 mg. histamine divided into 4 doses during the 12 hours previous to glucose	0.116	0.356	0.221	0.156	60.0	Toxic

obviously toxic, as was confirmed by the marked increase in the serum nonprotein nitrogen. There were no evidences of dehydration as measured by the percentage of corpuscles and the concentration of the serum protein except with puppy 6, in which the percentage of corpuscles increased from 52 to 70 per cent.

The Effect of a Chemical Toxin.—The results recorded in tables 2 and 6 illustrate quite clearly that a chemical toxin such as histamine may definitely impair the ability of the organism to remove injected glucose from the blood stream. The degree of toxicity as observed clinically

TABLE 3.—*The Effect of Dehydration in Puppies on the Blood Sugar Values Found Before and After the Administration of 10 cc. Per Pound of Body Weight of a 10 Per Cent Solution of Glucose*

Puppy No.	Puppy Weight	Substance of Treatment Given	Blood Sugar Before Administration of Glucose, per Cent	Blood Sugar ½ Hour After, per Cent	Blood Sugar 1 Hour After, per Cent	Blood Sugar 1½ Hours After, per Cent	Non-protein Nitrogen in Serum, Mg. per 100 Cc.	Serum Protein Determined by Kjeldahl per Cent	Condition of Dog
10	11	In cage for 46 hours; temperature of cage 35 C.; no food or water	0.103	0.176	0.138	0.098	50	6.2	Toxic; lost 2 pounds
11	10	In cage for 45 hours; temperature of cage 35 C.; no food or water	0.142	0.410	0.317	0.252	100	8.2	Very toxic
12	15	In cage for 4 hours a day for 3 days; temperature of cage 34 C.; no food or water	0.109	0.323	0.205	0.135	30	7.0	Apparently normal; lost 3 pounds
13	9	Same as puppy 12.....	0.078	0.400	0.295	0.230	50	7.2	Toxic; lost 1½ pounds
15	13	Same as puppy 12.....	0.100	0.240	0.206	0.156	28	6.1	Fairly lively; lost 3 pounds
Average values found in 6 normal puppies.....							18	...	4.7

TABLE 4.—*The Effect of the Previous Oral Administration of Magnesium Sulphate, Jalap and Castor Oil to Puppies, on the Blood Sugar Values Found Before and After the Administration of 10 cc. Per Pound of Body Weight of a 10 Per Cent Solution of Glucose*

Puppy No.	Puppy Weight	Substance Given	Blood Sugar Before Administration of Glucose, per Cent	Blood Sugar ½ Hour After, per Cent	Blood Sugar 1 Hour After, per Cent	Blood Sugar 1½ Hours After, per Cent	Condition of Dog
16	14	1 ounce of magnesium sulphate by mouth twice a day for 3 days	0.142	0.220	0.135	0	Profuse diarrhea; otherwise normal
46	8	1 ounce of magnesium sulphate by mouth twice a day for 2 days	0.141	0.320	0.185	0.125	No diarrhea; vomited; somewhat toxic
49	7	Same as puppy 46.....	0.115	0.360	0.250	0.188	No diarrhea; vomited; somewhat toxic
50	13	5 drams of powdered jalap by mouth divided into 5 doses during previous 60 hours	0.156	0.248	0.190	0.154	Profuse diarrhea; toxic
51	12	Same as puppy 50.....	0.106	0.262	0.170	0.115	Diarrhea; very slightly toxic
9	10	½ ounce castor oil by mouth 3 times a day for 2 days	0.120	0.292	0.173	0.120	No diarrhea; very slightly toxic
47	13	10 cc. paratyphoid-typhoid vaccine subcutaneously	0.108	0.261	0.122	0.095	Very slightly toxic

and as indicated by the concentration of the serum nonprotein nitrogen apparently influences the extent of the impairment to some degree. No essential change occurred in the concentration of the serum protein, so there is no evidence of dehydration.

The Effect of Dehydration.—We found it very hard to produce a dehydration without the development of intoxication. As indicated in table 3, the puppies were definitely dehydrated, as evidenced by their clinical condition and the concentration of the serum protein. Puppies 10, 11 and 13 showed clinical evidences of intoxication as well as dehydration. Although there was very little clinical evidence of intoxication in puppies 12 and 15, the serum nonprotein nitrogen was about 50 per cent greater than the amount usually found by us in normal puppies. Even allowing for an increased concentration of the blood, this would indicate some degree of intoxication. In view of this we cannot

TABLE 5.—*The Effect of an Elevation of Temperature in Puppies on the Blood Sugar Values Found Before and After the Administration of 10 cc. Per Pound of Body Weight of a 10 Per Cent Solution of Glucose. The Elevation of Temperature Occurred a Few Hours After the Administration of Diphtheria Toxin Before the Development of Toxic Symptoms*

Puppy		Temperature of Dog, Degrees F.	Blood Sugar Before Administration of Glucose, per Cent	Blood Sugar ½ Hour After, per Cent	Blood Sugar 1 Hour After, per Cent	Blood Sugar 1½ Hours After, per Cent	Nonprotein Nitrogen in Serum, Mg. per 100 Cc.
No.	Weight						
3	8	105.2	0.131	0.096	0.122	0.128	12.0
4	12	105.0	0.103	0.080	0.106	0.115	12.7
5	15	102.2	0.103	0.083	0.120	0.112	18.1

TABLE 6.—*The Average of the Values Recorded in Tables 1 to 4. For Comparison, Average Values Obtained with Infants Are Given*

No. of Puppies Used	Substance or Treatment Given	Blood Sugar Before Administration of Glucose, per Cent	Blood Sugar ½ Hour After, per Cent	Blood Sugar 1 Hour After, per Cent	Blood Sugar 1½ Hours After, per Cent
10	Normal	0.092	0.155	0.098	0.092
3	Diphtheria toxin subcutaneously	0.106	0.351	0.219	0.162
5	Histamine subcutaneously	0.106	0.338	0.220	0.151
5	Dehydration	0.106	0.312	0.232	0.174
3	Magnesium sulphate by mouth	0.132	0.300	0.190	0.155
2	Powder jalap by mouth	0.131	0.255	0.180	0.134
1	Castor oil by mouth	0.120	0.292	0.173	0.120
Normal infants		0.085	0.282	0.120	0.078
Infants with infections		0.085	0.370	0.260	0.185
Infants with diarrhea		0.075	0.360	0.255	0.170
Infants with acute intestinal intoxication		0.085	0.380	0.300	0.230

conclude that dehydration, per se, produces an impairment in the ability of the animal to remove injected glucose from the blood stream. It seems reasonable, however, in view of the similar results obtained with a toxemia or intoxication produced by both a bacterial and chemical toxin, to conclude that the impairment in the ability of the animal to remove injected glucose from the blood stream was the result of the intoxication produced by dehydration.

Beck [3] has shown that starvation in diabetes lowers the carbohydrate tolerance. Teetsu,[4] working in MacLeod's laboratory, also found that starvation in rabbits lowers the carbohydrate tolerance. As our dehydrated puppies received no food for from two to four days, this period of starvation may have had some influence on our results.

The Effect of Some Laxative Drugs.—An attempt was made to produce diarrhea by the administration of magnesium sulphate, powdered jalap and castor oil. The puppies were all on an ordinary diet with plenty of water. Magnesium sulphate was administered to three as indicated in table 4. Only the first puppy developed diarrhea. The other two appeared somewhat toxic. In all three there was an impairment of their ability to remove injected glucose from the blood stream. A similar result was obtained with jalap administered to two puppies and with castor oil to one puppy. These last three dogs appeared somewhat toxic, but only the first two developed diarrhea. There is no definite evidence from these experiments that diarrhea, per se, causes any impairment of the ability of the animal to remove injected glucose from the blood stream. The first puppy (puppy 16) developed a profuse diarrhea and showed no evidence of an intoxication, but in view of the fact that a mild degree of intoxication may not evidence itself clinically, we cannot rule out this effect. We may conclude, however, that an intoxication produced by the administration of magnesium sulphate, jalap or castor oil, whether accompanied by diarrhea or not, impairs the ability of the animal to remove injected glucose from the blood stream at the normal rate.

COMMENT

The observation which prompted this investigation was that infants with diarrhea, infections and acute intestinal intoxication could not remove injected glucose from the blood stream at the normal rate.[1] Is this impairment due to: (1) The elevation of temperature which almost invariably accompanied these conditions; (2) to the diarrhea; (3) to the toxemia or intoxication whether of bacterial or chemical origins, or (4) to dehydration?

During our studies on the carbohydrate metabolism of infants, we encountered only one infant with an elevation of temperature who removed injected glucose from the blood stream more rapidly than normal. This occurred in an infant who developed the elevation of temperature just one or two hours before the glucose was given. This observation and the results recorded in table 5 would indicate that elevation of temperatures, if not accompanied by intoxication, not only

3. Beck, W.: Ztschr. f. Klin. Med. **98**:465, 1924.
4. Personal communication.

does not impair the ability of the body to remove injected glucose but also causes a more rapid removal than normally occurs.

We were not able definitely to produce diarrhea without toxic symptoms, so there is no evidence from the present work that diarrhea per se affects the carbohydrate metabolism. We have, however, shown quite clearly that a bacterial toxin, a chemical toxin and the toxic condition caused by dehydration results in a definite impairment in the ability of the animal to remove injected glucose from the blood stream. We have also found that the toxic condition produced by such diverse substances as magnesium sulphate, jalap and castor oil results in a similar impairment. It would appear, therefore, that the impairment of the ability of the animals to remove injected glucose from the blood stream at the normal rate is the result of an intoxication, irrespective of the original agency causing this condition.

To go a step further, how does the toxemia produce this impairment in the carbohydrate metabolism? It might interfere with the production of insulin, and it probably does to a degree, but still the administration of insulin to infants with infections or intoxication produced very little effect.[1] This would indicate that a lack of insulin is not primarily the cause of this disturbance in the carbohydrate metabolism.

It has been found by Cajori, Crouter and Pemberton [5] that a diminished blood flow to the muscles may result in a delay in the removal of glucose from the blood stream. It is conceivable that in the conditions developed in the present experiments there is a marked decrease in the peripheral blood flow, and that this might account for the delay in the removal of the glucose from the blood stream. It has been observed by Utheim,[6] however, that the peripheral blood flow of marantic infants is markedly reduced, yet in these infants we found that injected glucose was removed from the blood stream with the normal rapidity.[7] Consequently, we do not believe that a diminished blood flow to the muscles is the important factor in our observations.

Is the disturbance due to an increased activity of the glycogenolytic function of the body; that is, is there an increase in the amount of glucose formed from the glycogen stores and subsequently discharged into the blood stream? O'Neill, Manwaring and Moy [8] showed that glycogen disappears almost quantitatively from the canine liver during the first

5. Cajori, F. A.; Crouter, C. Y., and Pemberton, R.: J. Biol. Chem. **66**:89, 1925.

6. Utheim, K.: Studies of Blood and Its Circulation, Am. J. Dis. Child. **20**:366 (Nov.) 1920.

7. Tisdall, F. F.; Drake, T. G. H., and Brown, A.: Carbohydrate Metabolism of Marantic Infant, Am. J. Dis. Child. **30**:829 (Dec.) 1925.

8. O'Neill, F. I.; Manwaring, W. H., and Moy, H. B.: Quantitative Changes in Hepatic Glycogen in Anaphylactic Shock, J. A. M. A. **84**:1102 (April 11) 1925.

fifteen minutes of anaphylactic shock. It was found by Chambers and Thompson [9] that the liver glycogen decreased nearly 80 per cent, and the heart and skeletal muscle glycogen about 60 per cent during the first fifteen minutes after the intravenous injection of histamine in dogs. At the same time, a marked increase occurred in the concentration of blood sugar. Similarly, a very rapid increase in the blood sugar concentration of dogs with anaphylactic shock was noted by McCullough and O'Neill,[10] while Hirsch and Williams [11] and Zeckwer and Goodell [12] with rabbits, and Zung and La Barre [13] with guinea-pigs, made the same observation. Menten and Manning [14] and Zeckwer and Goodell [15] found an increase in the blood sugar concentration as a result of the intravenous injection of certain killed bacteria. It is obvious that with the conditions produced by these investigators there is a mobilization of glycogen, and a resultant temporary rise in the blood sugar concentration. The fact that the initial blood sugar concentration before the injection of glucose was higher in all our treated animals than that found in our normal animals (table 6), would indicate that an increased glycogenolytic activity may be a factor in the production of our results.

It has been shown by Sanger and Hun,[16] by Linder, Hiller and Van Slyke,[17] and by others that only about 20 per cent of ingested glucose is utilized in from two to three hours following the ingestion. This indicates that the greater part of the glucose is stored. In our experiments the toxic condition developed slowly, so sufficient time elapsed to allow the development of a general impairment of the activity of all the cells of the body. We must therefore consider the possibility of an impairment in the ability of the organism to store the administered carbohydrate as another factor in the production of our results.

SUMMARY

1. The subcutaneous administration of large doses of a bacterial toxin (diphtheria toxin) in young dogs definitely impairs the ability of the animal to remove injected glucose from the blood stream at the normal rate.

2. The subcutaneous administration of large doses of a chemical toxin (histamine) in young dogs definitely impairs the ability of the

9. Chambers, E. K., and Thompson, K. W.: J. Infect. Dis. **37**:229, 1925.
10. McCullough, M., and O'Neill, F. I.: J. Infect. Dis. **37**:225, 1925.
11. Hirsch, E. F., and Williams, J. L.: J. Infect. Dis. **30**:259, 1922.
12. Zeckwer, I. T., and Goodell, H.: J. Exper. Med. **40**:57, 1925.
13. Zung, E., and La Barre, J.: Compt. rend. Soc. de biol. **91**:121, 1924.
14. Menten, M. L., and Manning, H. M.: J. M. Res. **44**:675, 1924.
15. Zeckwer, I. T., and Goodell, H.: J. Exper. Med. **42**:43, 1925.
16. Sanger, B. J., and Hun, E. G.: Glucose Mobilization Rate in Hyperthyroidism, Arch. Int. Med. **30**:397 (Sept.) 1922.
17. Linder, G. C.; Hiller, A., and Van Slyke, D. D.: J. Clin. Investigation **1**:247, 1925.

animal to remove injected glucose from the blood stream at the normal rate.

3. The rapid production of dehydration in young dogs definitely impairs the ability of the animal to remove injected glucose from the blood stream at the normal rate.

4. The oral administration of large doses of magnesium sulphate, jalap and castor oil definitely impairs the ability of the animal to remove injected glucose from the blood stream at the normal rate.

5. The impairment in the ability of the animal to remove injected glucose from the blood stream at the normal rate is not an effect of the substance given or of the dehydration produced, but is associated with a more profound functional derangement of the cells commonly called intoxication.

6. This intoxication may produce its effect on the sugar tolerance values either by an increase in the glycogenolytic function of the body or by an impairment of the ability of the organism to store carbohydrate.

7. Elevation of temperature without any evidence of intoxication not only does not impair the ability of the animal to remove injected glucose from the blood stream, but also, apparently, causes its removal at a more rapid rate than normal.

ASTHMA IN CHILDREN

II. THE INCIDENCE AND SIGNIFICANCE OF ECZEMA, URTICARIA AND ANGIONEUROTIC EDEMA *

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Czerny expressed the opinion twenty years ago that eczema in breast-fed infants was due to a disturbed fat metabolism. He therefore included these cases in his so-called exudative diathesis group. Since then, many differing theories have been advanced regarding the etiology of eczema, urticaria and angioneurotic edema, each finding a measure of support. It was not known until the introduction of the protein skin tests that a definite relation between protein sensitization and these dermatoses existed. This was established by the work of Blackfan,[1] Talbot,[2] Strickler,[3] White,[4] Schloss,[5] O'Keefe,[6] Shannon [7] and others.

The earlier investigators made the clinical observation that children with eczema were especially prone to recurrent attacks of so-called "non-infective rhinitis" and "bronchitis" or bronchial asthma. Holt [8] remarked that asthma following eczema in children was not infrequent. Schloss [5] stated that "a good proportion of infants or young children with bronchial asthma had eczema during infancy." Blackfan [1] said that patients who

* Received for publication, Aug. 5, 1926.

* From the Children's Asthma Clinic, Mt. Sinai Hospital.

1. Blackfan, K. D.: Cutaneous Reaction from Proteins in Eczema, Am. J. Dis. Child. **11**:441 (June) 1916.

2. Talbot, F. B.: Idiosyncrasy to Cow's Milk. Its Relation to Anaphylaxis, Boston M. & S. J. **175**:409 (Sept. 21) 1916; Relation of Food Idiosyncrasies to Diseases of Childhood, ibid. **179**:285 (May 29) 1918.

3. Strickler, A.: Anaphylactic Food Reactions in Skin Diseases with Specific Reference to Eczema, New York M. J. **104**:198 (July 29) 1916; Strickler, A., and Goldberg, J. M.: Anaphylactic Food Reactions in Dermatology, J. A. M. A. **66**:249 (Jan. 22) 1916.

4. White, C. J.: The Treatment of Eczema in Childhood, Boston M. & S. J. **178**:5 (Jan. 3) 1918.

5. Schloss, O.: Allergy in Infants and Children, Am. J. Dis. Child. **19**:433 (June) 1920.

6. O'Keefe, E. S.: The Relation of Food to Infantile Eczema, Boston M. & S. J. **183**:569 (Nov. 11) 1920; Eczema in Breast-Fed Baby and Protein Sensitization, ibid. **185**:194 (Aug. 18) 1921.

7. Shannon, W. R.: Demonstration of Food Proteins in Human Breast Milk by Anaphylactic Experiments on Guinea-Pigs: Their Probable Relationship to Certain Diseases of Nursing Infants: A Preliminary Report, Am. J. Dis. Child. **22**:223 (Sept.) 1921; Eczema in Breast-Fed Infants as a Result of Sensitization to Foods in the Mother's Dietary, ibid. **23**:392 (May) 1922.

8. Holt, L. M.: The Diseases of Infancy and Childhood, New York, D. Appleton & Co., 1919.

were unable to take different foods on account of urticaria, angioneurotic edema and asthma nearly always gave a history of eczema in early life. Unfortunately, these workers, with the exception of Moro and Kolb,[9] did not determine what proportion of infants with eczema subsequently developed asthma. Moro and Kolb [9] observed eighty-six infants with eczema over periods ranging from three and one-half to eleven years, and found the incidence of asthma to be 2.3 per cent. Almost without exception, only food proteins were used by those workers who employed the sensitization tests. The scope of their work was therefore limited. This study was undertaken to determine the relation between these dermatoses and asthma, indirectly, from their incidence in a group of children with asthma.

The material for this study consisted of 100 children with asthma,[10] ranging in age from 6 months to 14 years. These children were tested not only with food proteins, but also with pollen, dust and bacterial proteins. Seventy-nine per cent were protein sensitive, and 21 per cent nonprotein sensitive. Of these 100 cases, thirty-one presented a history of, or an existing, dermatosis. Of these thirty-one patients, twenty-two were protein sensitive and nine were nonprotein sensitive. The analysis of the cases showing dermatoses will be taken up under these two divisions.

PROTEIN SENSITIVE GROUP

Of the seventy-nine children with allergic asthma, eighteen (or 22 per cent) had eczema, two (or 2.5 per cent) had urticaria and two (or 2.5 per cent) had angioneurotic edema, a total incidence of 27 per cent with dermatoses.

Table 1 shows each case with the number of positive reactions in the various food and inhalant groups, the age of onset of eczema, urticaria or angioneurotic edema, together with the duration of the skin condition and the age when first observed.

In the seventy-nine children with allergic asthma, there were 274 positive cutaneous food reactions; in the twenty-two with dermatoses (table 1) there were 153 food reactions—about 56 per cent of the total. This incidence is interesting and requires clinical interpretation.

An analysis of table 1 shows:

1. Six patients reacting negatively to food proteins.
2. Six patients reacting to a total of nine food proteins, or from one to two per patient.

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9. Moro, E., and Kolb, L.: Ueber das Schicksal von Ekzemkindern, Monatschr. f. Kinderh. **9**:428, 1910.

10. Peshkin, M. M.: Asthma in Children: I. Etiology, Am. J. Dis. Child. **31**:763 (June) 1926.

3. Ten patients reacting to 144 food proteins, or from seven to twenty-two per patient.

1. The six patients failing to give positive cutaneous food reactions also gave a negative history of food intolerance. The etiology of the dermatoses in this group could not be ascertained. Eczema commenced in four patients at from 2 weeks to 11 months of age, while the interval between the time of disappearance of the rash and the onset of eczema ranged from 1 to 7 years. In another patient, the eczema which

TABLE 1.—*Allergic Asthma in Children with a Past or Present History of Eczema, Urticaria or Angioneurotic Edema*

Food Group	1	2	3	4	5*	6	7	8	9	10	11	12*	13	14	15	16	17	18	19	20	21†	22†
Cereal grains...	0	0	4	0	1	0	1	2	0	0	3	1	0	1	8	1	0	0	0	0	0	4
Egg.....	0	0	0	0	0	0	0	3	0	0	3	0	1	0	3	3	0	0	3	0	0	0
Milk.....	2	0	1	0	0	0	0	1	0	0	0	0	2	0	2	0	0	0	1	0	0	4
Meat.....	5	0	0	0	0	0	0	4	1	0	0	0	1	0	0	0	1	0	5	0	0	1
Vegetable.....	0	0	5	0	0	0	0	2	0	1	2	1	2	0	8	2	0	0	2	0	0	0
Fish.....	9	0	0	0	0	0	0	4	6	0	4	0	11§	0	0	6	0	0	0	0	0	0
Fruit.....	0	0	0	0	0	0	0	0	0	0	0	0	1	1	0	0	1	0	0	0	0	1
Beverage.....	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	1
Nut.....	1	0	0	0	0	0	0	1	0	0	2	0	1	0	0	1	0	0	1	0	0	1
Total food reactions.....	17	0	10	0	1	0	1	17	7	1	14	2	19	2	22	14	2	0	12	0	0	12
Inhalant reactions.....	3	3	6	4	2	2	1	3	2	3	5	5	7	4	8	5	7	1	3	2	2	5
Pollen reactions	0	0	0	0	0	0	0	2	0	0	1	0	0	1	5	2	2	0	2	0	0	0
Total protein reactions.....	20	3	16	4	3	2	2	22	9	4	20	7	26	7	35	21	11	1	17	2	2	17
Age onset of skin lesion....	6m	11m	3m	2w	5	2m	2m	2m	3m	6w	2m	2	3m	3w	3m	2m	3m	2w	6w	2w	11	7
Duration of skin lesion....	6m	10m	9m	2	3m	P	9m	22m	9m	5m	P	C	6m	R	R	P	P	6m	8m	9m	2d	6m
Age onset of asthma.....	5½	6	4	9	1	6	1	2	4	1	6	2	3½	2½	1½	1½	2½	1½	2	4	9	3
Age first seen...	7½	12	4	13½	5	8	11	8	7	10	8½	6½	5½	7	4½	5½	9	3	3	9	12	6

* Urticaria.
† Angioneurotic edema.
§ Includes four shellfood proteins.
The figure above each column indicates the number of the case. The figures in each column indicate the number of proteins. The food groups are listed in a recent publication. The cereal grains include the split proteins of whole wheat. The egg group includes eggwhole, eggwhite and eggyolk. The milk group includes whole milk, casein, lactalbumin and cheese. M = month; w = week; d = day. The age not designated is interpreted in terms of years. P = persistent rash; R = recurrent rash; C = contact with a live rabbit.

commenced at 2 months of age was still present when the child was first observed at 8 years, two years after the onset of asthma. One patient, at the age of 11 years, had angioneurotic edema of unknown etiology two years after the onset of asthma.

2. In this group of six patients, there was a maximum of two food reactions per patient. Two of these patients had urticaria, the ages of onset of which were 5 and 2 years, while the age of onset of asthma was 1 and 2 years, respectively. The urticaria in one was caused by contact with a live rabbit, while in the other the cause was unknown. Four patients had eczema which commenced at from 3 weeks to 4 months of age. In two of these four patients, the interval between the disappearance of eczema and the age of onset of asthma was one and five months, respectvely. In one of the remaining two, eczema was persistent and

asthma set in one and one-fourth years after the appearance of the skin lesion; in the other, eczema was recurrent and asthma set in two and one-fourth years after the appearance of the rash. The ages at which these two cases were first observed were 9 and 7 years, respectively. There was no history of food intolerance in this group. Neither the eczema nor the asthma was relieved by the exclusion of these sensitizing foods or by other dietary regulation.

3. The third group, consisting of only 12.6 per cent of the entire allergic asthma series, accounted for 52 per cent of the total food sensitizations. These patients reacted to from seven to twenty-two foods each, or an average of 14.4 per patient.

Nine patients had eczema which commenced at from 2 weeks to 6 months of age. In five of these the eczema cleared up completely, and asthma developed in from one to six years after the disappearance of the rash. In two patients, the eczema was persistent, asthma commencing one and five years, respectively, after the onset of eczema. In another, the eczema was recurrent, asthma beginning one year after the onset of eczema. In the ninth case, eczema continued for twenty-two months and cleared up with the onset of asthma at 2 years. The tenth patient of this group developed angioneurotic edema four years after the onset of asthma.

Each of the patients of this group exhibited cutaneous hypersensitiveness to one or more biologic food groups, such as fish, meat, cereal grains, etc. They were the only patients in the entire asthma series who reacted to foods in this manner.

Only six patients reacted to fish. But in each case the reactions were to the biologic group. A history of intolerance to fish was obtained in four of these patients. In one, contact with fish always caused erythema and angioneurotic edema, while ingestion caused asthma (case 1). In another, the odor of fish caused nausea, while ingestion was always followed by vomiting, and the eating of salmon caused asthma (case 9). In a third patient, the ingestion of any kind of fish caused urticaria and exacerbation of the existing eczema, which was persistent (case 16). In the fourth patient, the ingestion of fish caused urticaria only (case 13). In this group of asthmatic children, the clinical manifestations of hypersensitiveness to fish were mainly referable to the skin as urticaria, erythema and angioneurotic edema. Among those patients who had "outgrown" the eczema, ingestion of, or contact with, fish did not cause a return of the eczema, but did produce secondary skin rashes. In those in whom eczema was persistent or recurrent, the ingestion of fish caused an exacerbation of the skin condition, but the elimination of fish from the diet did not relieve the eczema. From these observations it may be stated with reasonable definiteness that a child with asthma exhibiting

positive cutaneous reactions to fish as a biologic group will almost invariably give a history of having had eczema during infancy.

Seven patients reacted to one or more proteins of the meat group. All gave a negative history of intolerance. However, in the three patients who reacted to the entire meat group, the asthmatic symptoms were lessened after the exclusion of these foods from the diet. The eczema which commenced during infancy, and which was absent in all cases when these patients were first seen, was not observed to recur following the ingestion of the meats. It may be stated that children with asthma exhibiting positive cutaneous reactions to a biologic meat group will almost invariably give a history of eczema during infancy, that the eczema is rarely persistent, and that, once the eczema has cleared, the ingestion of the meats rarely causes any further skin disturbances.

Seven patients reacted to milk. Only two of these gave a history of intolerance. In one of these (case 13), ingestion caused urticaria, and in the other (case 15), merely a dislike for milk. Symptoms of intolerance were first manifested in these two patients at the weaning periods, 6 and 11 months of age, respectively. In one of these, milk subsequently played a prominent rôle in the onset of asthma at 1½ years of age. All of these children now drink from three to four glasses of milk daily without its causing any symptoms, in spite of the presence of positive skin reactions. Of this group, angioneurotic edema occurred in one patient after the onset of asthma, and in the remaining six, eczema commenced during infancy and prior to the onset of asthma. Three of these patients reacted in conjunction with the fish group.

Six patients reacted to egg as follows: five to eggwhole and eggwhite, and one to eggwhole only. Four of these patients also reacted to the fish group. Two of these four patients gave no history of intolerance to egg; in the third, egg caused only vomiting since infancy, and in the fourth, vomiting and asthma. The remaining two patients gave a history of persistent intolerance to egg, causing nausea in one and asthma in the other. It would appear that the existing clinical manifestations of hypersensitiveness to egg were mainly referable to the gastro-intestinal and respiratory tracts.

The cereal grains hold the same position, clinically, as milk. When these patients were first observed, they had already acquired a tolerance for these foods.

The remaining food groups, with the exception of nuts, assume a minor rôle in the causation of symptoms. Nuts cause asthma, or urticaria, or both.

In patients with a persistent or recurrent eczema, it was not uncommon to obtain a clinical history of alternating asthma and eczema. I

recall the case of a boy whose parents refused to employ the usual medication for the relief of the eczema because the clearing of the eczema was always followed by the more undesirable asthmatic symptoms. However, such a relation was not observed to occur in this case after the eczema was cleared by treatment with the mercury vapor quartz lamp.

THE COMPARATIVE INCIDENCE OF FOOD SENSITIZATION IN CHILDREN WITH ASTHMA GIVING A POSITIVE AND NEGATIVE HISTORY OF ECZEMA, URTICARIA OR ANGIONEUROTIC EDEMA

It has already been stated in the foregoing that 27 per cent of the children with allergic asthma accounted for 56 per cent of the food sensitizations. It was found that this group of 27 per cent was composed of children giving a positive history of eczema, urticaria and angioneurotic edema, and was referred to as the dermatoses group. Table 2 shows the number of children with asthma reacting to food proteins and the comparative incidence of the various food sensitizations in these children, arranged in two groups, according to whether they gave a positive or a negative history of dermatosis.

TABLE 2.—*The Comparative Incidence of Food Sensitizations in Children with Asthma Giving a Positive and Negative History of Eczema, Urticaria or Angioneurotic Edema*

	Sensitizations of Patients in Whom History of Dermatoses Was:					Patients in Whom History of Dermatoses Was:				
Food Group*	Positive	Negative	Total	Per Cent +	Per Cent —	Positive	Negative	Total	Per Cent +	Per Cent —
Fish	40	3	43	93	7	6	3	9	10	5
Meat	18	3	21	85	15	7	3	10	12	5
Milk	13	3	16	81	19	7	3	10	12	5
Egg	16	6	22	72	28	6	3	9	10	5
Wheat	26	31	57	45	55	10	20	30	18	36
Nut	8	10	18	44	56	7	6	13	12	10
Fruit	4	6	10	40	60	4	6	10	7	10
Vegetable	26	43	69	37	63	9	25	34	16	44
Beverage	2	16	18	11	89	2	16	18	3	28
Total	153	121	274	55.8	44.2	16	40	56	28	72

* The various food groups are explained in table 1.

It is of extreme interest to note (table 2) that 93 per cent of the reactions to fish were encountered in only 10 per cent of the children with allergic asthma, that meat accounted for 85 per cent, milk for 81 per cent and egg 72 per cent of the reactions. The remaining food groups have a much lower incidence and play a lesser rôle in the causation of symptoms. It therefore appears that the empiric restriction of fish, meat, milk and egg from the dietary of children with eczema as practiced by the clinician of former years was justified. None of the foods to which these children reacted was the sole factor in the causation of asthma. In fact, more than one half of the food sensitizations did not

enter into the etiology of asthma at any time, but were responsible for inducing the various skin manifestations of allergy, an observation different from that of Walker [11] and others.

THE RELATION BETWEEN THE AGES OF ONSET OF ASTHMA AND THE DERMATOSES

A striking observation in these patients with allergic asthma was the persistence with which eczema always commenced during infancy, and always preceded the onset of asthma. In eleven of the eighteen subjects, the time interval of freedom from eczema to the onset of asthma was from one to seven years. In one, the time interval was one month. In the remaining six patients in which the eczema was persistent or recurrent, one and one-fourth to six years elapsed before asthma set in.

The observation that the primary urticaria and angioneurotic edema occurred always after the onset of asthma is significant. In these cases the asthma preceded the skin lesions by from two to four years in three cases and was concurrent in the fourth. In this case exposure to a live rabbit was the causal factor. Urticaria and angioneurotic edema following eczema and preceding or following the onset of asthma was a common observation, but these cases were not included in this series because they were not the primary skin lesions. Urticaria and angioneurotic edema rarely occur as a primary disturbance in children with allergic asthma.

NONPROTEIN SENSITIVE GROUP

Twenty-one of the 100 cases of asthma studied were nonprotein sensitive. Five of these had urticaria and four eczema; none presented angioneurotic edema. The relation between the age of onset of asthma and urticaria and eczema in these cases is shown in table 3.

TABLE 3.—*The Relation Between the Age of Onset of Asthma and Urticaria and Eczema in Nonprotein Sensitive Asthma*

	Case Number								
	23*	24	25*	26	27*	28*	29*	30	31
Age at onset of skin lesion.....	5y	5m	9½y	21m	6w	6w	8m	18m	2¼y
Duration of skin lesion.....	1d	7m	5d	3w	3w	P	3d	4m	6m
Age at onset of asthma.....	4¾y	1y	8½y	17m	8m	4½y	4m	4y	1y
Age of patient when first seen..	5y	2¾y	9½y	1½y	13m	4¾y	8y	6y	20m

* Urticaria.
d indicates day; w, week; m, month; y, year; P, persistent rash.

Three of the five patients with urticaria developed the rash from three months to one year after the onset of asthma. The urticaria remained from one to five days only. In the other two cases urticaria preceded the onset of asthma by one-half and four and one-half years,

11. Walker, I. C.: Oxford Medicine, New York, Oxford University Press, 1920.

respectively. The urticaria lasted three weeks in the first case and was persistent in the second. The urticaria in three of these patients occurred during infancy. The etiology could not be determined.

Two of the four patients with eczema definitely developed the lesion during a course of roentgen-ray therapy for enlarged tracheobronchial glands. In these two cases (26 and 31), the eczema lasted three weeks and six months, respectively. Of the remaining two cases, in one the eczema preceded the asthma by seven months and cleared up when the asthmatic symptoms began; in the other, it preceded the asthma by one and one-half years and cleared up two years before asthma set in.

It can readily be observed that in children with asthma of the nonprotein sensitive group, urticaria and eczema do not have the same relationship to either their primary occurrence or the onset of asthma as in those of the protein sensitive group.

Primary urticaria was six times more frequent in the nonprotein sensitive group. Eczema was four and one half times more frequent in the protein sensitive group. It occurred in infancy in all the cases of the protein sensitive group, and in only one of the nonprotein sensitive group.

COMMENT

In the collation of the incidence of eczema, urticaria and angioneurotic edema in an additional 100 cases of asthma subsequent to this study, the incidence was approximately the same as in this series, except that in the nonprotein sensitive group the incidence of eczema was considerably less than in this series. This can be accounted for by the fact that among the four cases of this series, two were included in which the etiology definitely was roentgen-ray irritation. With these two cases excluded, the incidence of eczema in both series was practically the same.

The fact that primary urticaria occurred in 24 per cent of the nonprotein sensitive cases and in only 2.5 per cent of the protein sensitive group would seem to indicate that primary urticaria in children with asthma is most often of nonallergic origin. This appears to be the impression of many workers in this field.

Many children react to numerous food proteins without their having even been sensitized by ingestion to these foods. These patients usually react to foods in biologic groups. This seeming anomaly could be explained by the fact that most of these children were breast-fed, and their sensitization to these foods is probably acquired through the mother's milk. Another example of sensitization to biologic protein groups is found in children sensitive to pollens, insect borne as well as air borne, although they may never have been exposed to some of these.

Walker [11] stated that the same food protein caused both the eczema and the asthma when these conditions occurred in the same patient.

The observations in this study would indicate that Walker's conclusions were erroneous. In this series, the inhalant proteins were the cause of asthma in the majority of the cases, while the food proteins assumed a minor contributory rôle.

The fact that children with eczema do react to certain foods in biologic groups suggests the possibility of using this knowledge in the prevention of asthma. If an infant or young child with eczema is shown to be sensitive to a biologic food group, then this child should be tested further with pollen and inhalant proteins. From the observations in this study, a goodly proportion of these subjects will show sensitization to some of the other proteins which have a more direct bearing on the etiology of asthma. The elimination of these sensitizing substances may prevent the onset of asthma.

CONCLUSIONS

1. In a study of 100 consecutive cases of asthma, 22 per cent of the patients had eczema; 7 per cent, urticaria and 2 per cent, angioneurotic edema, a total incidence of 31 per cent. Urticaria and angioneurotic edema secondary to an eczema were not included in these figures. In seventy-nine of these patients who were protein sensitive, the incidence of eczema was 22 per cent; urticaria, 2.5 per cent and angioneurotic edema, 2.5 per cent. In twenty-one who were protein nonsensitive, the incidence of eczema was 19 per cent; urticaria, 24 per cent, and there was no angioneurotic edema.

2. The patients comprising the 27 per cent of the allergic asthma group accounted for 55.8 per cent of the total food sensitizations. Of these, 7.5 per cent gave no food reactions, another 7.5 per cent reacted only to one or two foods, while the remaining 12 per cent accounted for 52 per cent of the total food reactions. The last group always reacted to foods in biologic groups, and the dermatoses were always either eczema or angioneurotic edema. The patients with urticaria did not react to biologic food groups.

3. Eczema always commenced during infancy and always preceded the onset of asthma, varying from one to seven years. Angioneurotic edema was concurrent with, or set in after, the onset of asthma. Urticaria always occurred after the onset of asthma. This relation did not exist in the nonallergic group.

4. Patients with asthma exhibiting positive cutaneous food reactions whose eczema had cleared did not have a recurrence of eczema with the ingestion of these sensitizing foods. Ingestion of these foods, however, did cause urticaria or angioneurotic edema and occasionally asthma in some cases. Patients with persistent or recurrent eczema were not relieved of the skin condition by elimination of these foods from the

diet. Their ingestion, however, resulted in an aggravation of the eczema, especially in those who were sensitive to biologic food groups.

5. The foods responsible for the greatest number of skin reactions in the dermatoses group were fish, meat, milk and egg.

6. All children with asthma reacting to fish or meat as biologic groups invariably gave a history of eczema commencing during infancy.

7. The existing clinical manifestations of hypersensitiveness to fish were mainly referable to the skin, and were, for example, urticaria, erythema and angioneurotic edema. Hypersensitiveness to meat was manifested in the respiratory tract; to egg in the gastro-intestinal and respiratory tracts, and to milk, in the gastro-intestinal tract and the skin. The clinical manifestation of intolerance to the other foods, except nuts, were few and unimportant. Nuts caused symptoms referable to the skin and respiratory tract. None of these foods was the sole cause of asthma.

8. In infants and young children with eczema reacting to foods as a biologic group, it may be possible to prevent asthma by further tests and by elimination of the potential sensitizations which have a direct bearing on the etiology of asthma.

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DUODENAL ULCERS IN CHILDREN *

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The diagnosis of chronic peptic ulcer in children is rarely made except at operation or at necropsy. Proctor [1] collected from the literature nineteen obvious cases, with two that were questionable, and added reports of three others observed at the Mayo Clinic. Another case [2] was recently reported from our clinic, the diagnosis being confirmed by operation. Of the twenty-five cases now reported in the literature, five were diagnosed at necropsy, eleven at operation and nine clinically. Of the last mentioned group, several were confirmed at operation; in the other cases the patients were given medical treatment. Only five of the twenty-five cases were diagnosed at any time as duodenal ulcer. Ten cases occurred in boys and fifteen in girls.

Within the last year, three patients have been seen in the Stanford Children's Clinic, who, because of their clinical history, the roentgenologic observations and the prompt response to treatment, I believe were suffering from chronic duodenal ulcers.

CASE REPORTS

CASE 1.—*History.*—F. M., a boy, aged 9 years, of Italian ancestry, said that ever since he could remember, he had had pain in his stomach after eating and after exercise, but it had been worse for the two months preceding admission to the hospital. The pain occurred every day; nausea was present at times, but the patient had never vomited. No radiation of pain was noted. On questioning, the patient localized the "stomach" pain in the left upper quadrant and in the mid-epigastrium.

Physical Examination.—There was evidence of a rather severe chronic sinusitis. The first right upper molar was abscessed. There was tenderness in the left upper and left lower abdominal quadrants, with some muscle rigidity. Blood, urine and stool examinations were negative. The Wassermann test was negative, and the tuberculin test was slightly positive. The stomach contents showed 20 units of free hydrochloric acid, and total acidity of 44 units. At fluoroscopy, after a barium meal, the duodenal cap filled poorly. Films showed the cap fairly smooth close to the pylorus but, as was noted on repeated films, irregularity persisted about 2 cm. from the pylorus, with a region which tended to retain a small spot of barium at the uppermost portion of the duodenum. After giving atropine until a physiologic effect was evident, the same deformity of the bulb was noted fluoroscopically. Some of the films of the stomach, however, showed a bulb of normal appearance. An immediate examination of the stomach, repeated two days later without using atropine, showed the old defect in the duodenal bulb on the screen as well as on all the films (fig. 1). There was a small residue

* Received for publication, Aug. 28, 1926.

* From the Division of Pediatrics, Stanford Medical School.

1. Proctor, Oscar S.: Chronic Peptic Ulcer in Children, Surg. Gynec. Obst. **41**:63-69, 1925.

2. Dickey, Lloyd B.: Duodenal Ulcer, with Diverticulum, J. A. M. A. **86**: 815-816 (March 20) 1926.

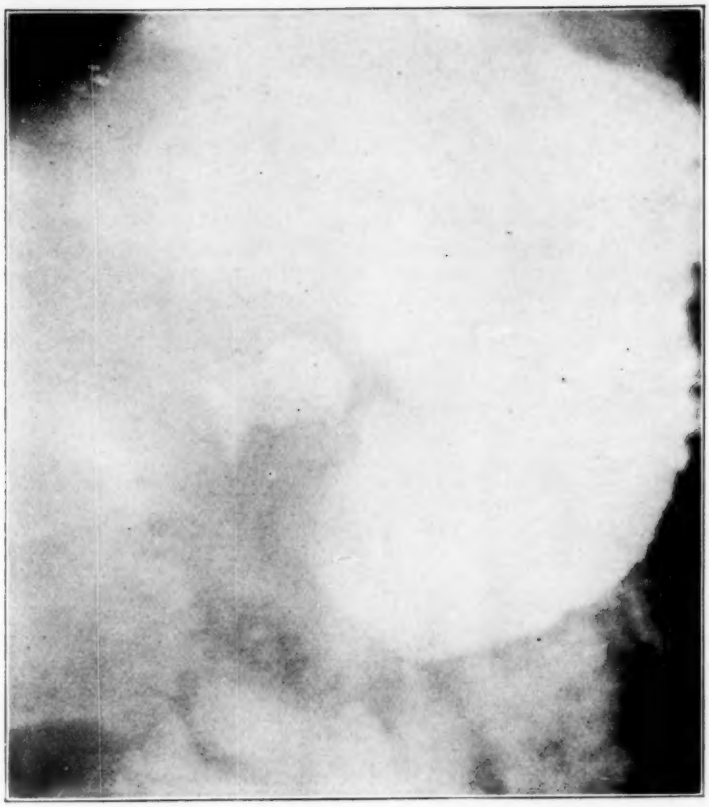

Fig. 1 (case 1).—Defect in duodenal bulb may be seen.

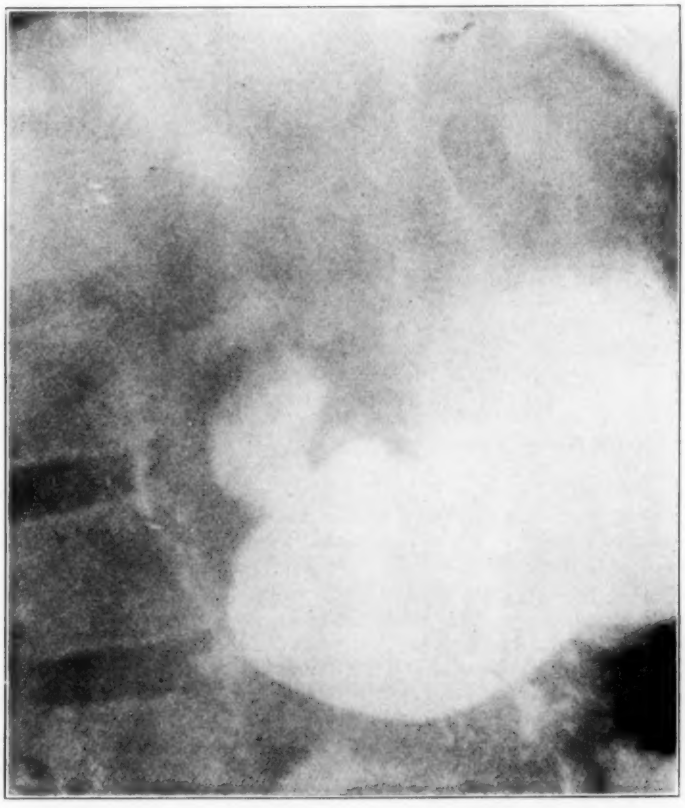

Fig. 2 (case 2).—The bulb had the appearance of being irregular and spastic.

of barium in the stomach six hours afterward. The gallbladder was normal in outline after tetraiodophenolphthalein had been given intravenously.

Course—On a modified Sippy diet the patient's pain and discomfort immediately disappeared, as did also the tenderness in the abdomen. Seven months later he was still entirely symptomless.

CASE 2.—*History.*—L. N., a Hungarian boy, aged 11 years, 7 months, since the age of 5 years had had attacks of pain which he localized in the right lower quadrant and in the midepigastrium. The pain, which did not radiate, usually

Fig. 3 (case 2).—Lower left portion of bulb appeared irregular, and suggestive of an ulcer crater.

came on about one hour after meals, and was associated with nausea but never with vomiting. It often awakened him at night. From the age of 5 to 6, he frequently passed blood in the stools, which were dark. At the age of 8, some stools were tarry in character. The epigastric pain was sometimes relieved by soup and milk, and made worse by meat. The effect of soda had not been tried.

Physical Examination.—The tonsil remnants were cryptic and reddened, with much lymphoid tissue in the posterior pharynx. Most of the cervical lymph nodes were palpable. There was definite tenderness to pressure in the right lower quadrant, and also just to the right of the umbilicus. Blood, urine and stool

examinations were normal. The Wassermann test was negative; the tuberculin test was positive. The stomach contents showed 50 units of free hydrochloric acid and 10 of combined acid. Fluroscopically, after a barium meal, the pyloric antrum was spastic and the emptying of the stomach was somewhat delayed. Later, by manipulation, the stomach was made to empty and to fill the duodenum. The bulb, however, filled only momentarily, and always had the appearance of being irregular and spastic (fig. 2). Reexamination of the patient, after atropine had been given until a physiologic effect was evident, showed the duodenal bulb much larger and better filled. The outline of the bulb, however, was not perfect,

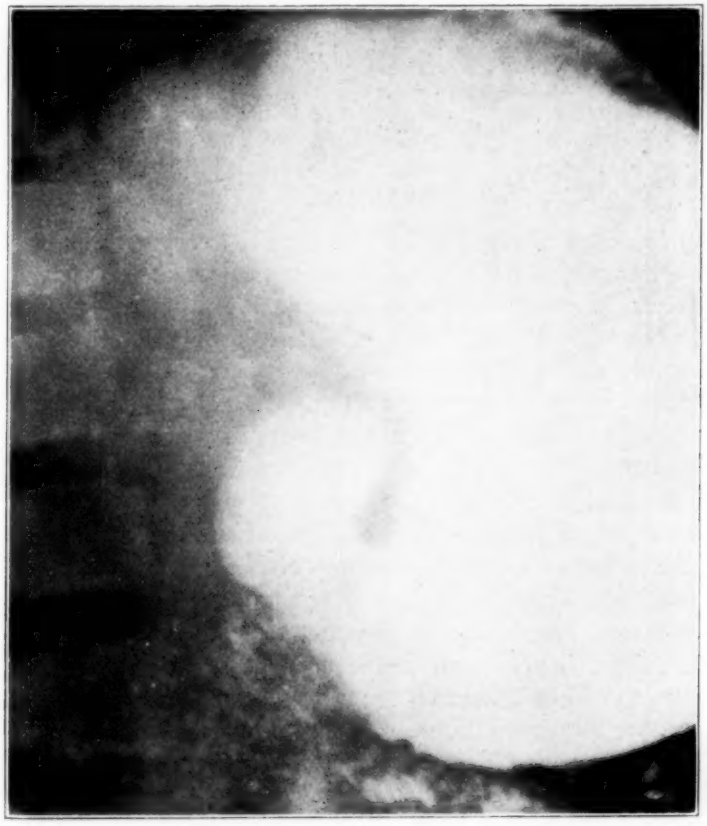

Fig. 4 (case 3).—Bulb with an outline suggestive of ulcer.

the lower left portion appearing irregular and being suggestive of an ulcer crater (fig. 3). A gallbladder, normal in outline, was visualized after tetraiodophenolphthalein had been given by mouth.

Course.—The patient, on a form of the Sippy diet, almost immediately became free from symptoms. He volunteered the information that he slept better than at any time he could remember. He is symptomless at the time of writing, about six months after the beginning of treatment.

CASE 3.—*History.*—J. L., a French boy, aged 8 years, four months before admission began to complain of abdominal pain, which he localized in the right

lower quadrant. He complained several times a week of pain, which was relieved by fluids such as tea. No relation of pain to meals was noted.

Physical Examination.—The tonsils were hypertrophied, and there were a few carious teeth. There was definite tenderness in the epigastrium, with no rigidity. Blood and urine were normal. At one examination, the stool showed cysts of *Giardia intestinalis*. The Wassermann test was negative; the tuberculin test was positive. The stomach contents showed 22 units of free hydrochloric acid, and 9 of combined acid. Roentgenograms, taken after tetraiodophenolphthalein was given by mouth, showed a gallbladder normal in outline. By fluoroscopy, after a barium meal, the stomach appeared high and tonic, and peristalsis was active. It did not begin to empty promptly, and emptying proceeded rather slowly. The duodenal bulb never appeared well filled, and usually only as a small black dot. The films also showed a bulb never well filled, and with an outline suggestive of ulcer (fig. 4).

Course.—The patient was placed on a form of the Sippy diet, and immediately became free from symptoms, continuing so up to the time of writing, five months after the beginning of treatment.

COMMENT

From the cases reported in the literature, it is evident that epigastric pain is a common, if not the most common, symptom and complaint of peptic ulcer in children. Some abdominal discomfort is practically always present. Although children do not always localize pain correctly, tenderness in the midepigastrium is usually elicited. The pains are usually, but not always, related to meals, and are relieved by food. Pains during the night are frequent. There may or may not be a seasonal incidence.

Nausea and vomiting are common complaints Nausea was a symptom in two of the foregoing cases, but vomiting was not present in any of the patients. There was a history of bleeding in only one of these cases, but it was noted by Proctor as occurring in about 40 per cent of those reported. Hyperchlorhydria is not common.

What, then, should arouse suspicion of peptic ulcer in children, and what facts should lead one to make a diagnosis before seeing the lesion after opening the abdomen?

As Proctor writes, "The most important single factor in the diagnosis is the realization that chronic peptic ulcer occurs in children." The histories of 2,000 patients at the Mayo Clinic treated for chronic peptic ulcer were reviewed, and it was found that forty-two of these presented symptoms dating back to childhood. This incidence of slightly more than 2 per cent points to the probable frequency with which chronic peptic ulcer in children is overlooked. Chronic abdominal pain, not easily explainable on some other basis, should lead to the suspicion of peptic ulcer, and to further investigation.

In most of the cases reported roentgenograms were not made. In those subjected to this kind of examination, lesions near the pylorus were noted.

Most conditions known as peptic ulcer were found or diagnosed probably rather late in the disease; hence the effect of medical treatment is not known with certainty, but there is no reason to believe that it would be different from that given to adults.

It is probable that a diagnosis of chronic peptic ulcer in children is warranted with the following facts: A history of chronic abdominal pain with epigastric tenderness, usually accompanied by nausea or vomiting, and these symptoms not explainable on another basis, with roentgenologic evidence of a lesion near the pylorus, and with immediate and complete relief of symptoms on a diet for patients with ulcer.

SUMMARY AND CONCLUSIONS

Three cases of what apparently are duodenal ulcers in children are reported, all of which gave roentgenologic evidence pointing to this lesion.

All of the patients were remarkably relieved on a diet for patients with ulcer.

If a careful investigation of chronic abdominal complaints in children is made, which includes roentgenologic studies, it is probable that the incidence of chronic peptic ulcer in children will be found to be greater than has hitherto been believed, and that these patients may be easily relieved of rather distressing symptoms.

ACTINOBACILLUS MENINGITIS*

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The actinobacillus has received little, if any, consideration as an agent of human infection, though the literature abounds with reports of such infections by other organisms of *Actinomycetaceae*. From the first reports of the "classical actinomycosis" down through the recent cases largely attributed to the streptothrices, the confusion in nomenclature makes any definite classification difficult. The terms most frequently employed are *Actinomyces, Cohnistreptothrix, Nocardia, Discomyces,* and *Streptothrix,* with the name *Actinobacillus* appearing only once.[1] In the last "Manual of Determinative Bacteriology" of the Society of American Bacteriologists, all of these names seem to be included in the genus *Actinomyces,* except *Actinobacillus* which is a genus by itself.[2] We believe the case which we are reporting here is a true infection with *Actinobacillus.*

CASE REPORT

L. O. M., a boy, aged 11 months, was seen at his home, Nov. 8, 1925, and was admitted to Saint Mary's Hospital, November 9.

Family History.—The father, aged 30, is living and well, but not robust. The mother, aged 26, is living and well; though fairly robust, she suggests a possibility of a sluggish endocrine system. There have been two previous pregnancies, one resulting in a miscarriage in 1921; the other, in a stillborn boy in January, 1923. The patient referred to here was born Nov. 22, 1924. The family physician says there is no syphilis in the family and there is no history of neurologic disease in any member of the family of either parent. They have always lived in their present locality, and have never been out of the United States.

Past History.—Birth was normal, and occurred at full term. The patient was a very large baby; at the age of 6 months he weighed 24 pounds (10.9 Kg.). When 5 or 6 weeks old, he "had a bad cold, with threatened pneumonia," but recovered after two weeks of illness. Otherwise, he had never been sick before the onset of the present illness. He was breast fed for ten months, then he was started on artificial feeding, and for a week was given part cow's milk and part breast milk; this was followed by feeding cow's milk entirely.

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* Received for publication, Aug. 5, 1926.

* From Saint Mary's Hospital, and the Laboratory of the City Board of Health.

1. Pinoy and Ravaut: Actinobacillose a forme méningée observée a Paris chez un Argentin, Presse méd. **50:** (Jan. 21) 1911.

2. Claypole: J. Exper. Med. **17:**99, 1913. Bell, H. H.: Infection of Meninges and Lungs by a Species of Actinomyces, J. Infect. Dis. **30:**99 (Jan.) 1922.

Present Illness.—About Oct. 29, 1925, the patient developed a "cold," with a slight cough during the first days. This ceased suddenly and all symptoms of the cold are said to have abated at the same time. With the cold, or perhaps just prior to it, diarrhea began, which lasted a week, and on November 5 the family physician was called. Lactic acid milk was advised; on November 6 this was changed to condensed milk, and such feeding was continued until admission to the hospital, November 9. Elevation of temperature was present with the cold and throughout the entire course of the disease.

On November 7, it was noted that the patient was more drowsy and sluggish than usual, and did not notice anything happening around him. He would make no effort to play with objects which had previously attracted his attention, and did not even appear to see them. He cried out when touched or moved. The temperature did not seem to be any higher than before.

On November 8, we saw the child for the first time, in consultation, and noted the following:

Physical Examination.—The patient was large, showing signs of recent loss of weight. He lay quietly in bed taking no notice of anything which did not touch him, though he seemed able to see objects held before him. He cried as if in pain when moved, and especially if his head was moved. There was some stiffness of the neck, otherwise he was rather flaccid.

The skin was smooth, moist and flabby, showing a loss of subcutaneous fat, especially over the abdomen. The anterior fontanel was large, tense, bulging and pulsating. The pupils of the eyes were equal and reacted to light. The eyegrounds could not be seen distinctly. Mild photophobia was present. The drums of the ears were normal. There was no evidence of any mastoid disease. Four sound teeth and normal gums were noted. The throat was normal. There was no glandular enlargement. The heart and lungs were normal. The abdomen was soft and lacked tone, but no masses or tenderness were present. The extremities were flaccid; the reflexes were difficult to obtain. Kernig's sign was not present. There was no opisthotonus. The tentative diagnosis was encephalitis.

On admission to the hospital, the physical observations were the same as on the previous afternoon. Roentgenograms of the skull showed widely separated sutures, with bulging fontanel; the sella turcica was apparently normal, with no symptoms to suggest a tumor. A spinal puncture was made, and about 30 mg. of fluid was removed, which presented a turbid appearance; later on, several ragged threads of dense clot festooned from side to side in the tube. The fluid was too old for cytologic examination when received at the laboratory, though the smear showed a large preponderance of polymorphonuclear leukocytes. The patient seemed brighter during the remainder of the day. On the morning of November 10, a second lumbar puncture was made, and 30 mg. of fluid was removed. This fluid showed no noticeable difference from that of the previous day. Improvement was less marked and of shorter duration. During the night there were twitchings, and bromides were given to prevent convulsions. November 11, the reflexes were exaggerated, a slight Kernig's sign was present, and the fontanel was bulging again. Spinal puncture was again made, the report on which may be found in the laboratory examination. Punctures were not made on November 12 or 13. The patient continued comatose and was kept under the influence of bromides. November 13, at 6 a. m., the infant died. Permission for necropsy was not obtained.

LABORATORY EXAMINATION

Spinal Fluid.—The first puncture on November 9 yielded slightly cloudy fluid, which by the time it reached the laboratory had several filamentous coagula festooned from the surface of the liquid to the sides of the tube below. Accurate cell count was impossible, but there was a striking increase in leukocytes, apparently all polymorphonuclears, some large and degenerate. Ziehl-Neelson stain washed all material from the slides. Gram's stain showed short bacilli, some very slender, others thick, some fusiform and slightly curved. Occasionally, a bacillus with a swollen or clubbed end was found. There were also many coccoid forms,

and a few diplococci which seemed encapsulated. All were gram-negative. Cultures made in beef extract broth enriched with blood showed good growth, which will be described later.

The fluid from the puncture of November 11 showed marked increase in cloudiness. Cell count showed 2,210 cells, 98 per cent polymorphonuclear leukocytes. Gram's stain showed large and small gram-negative organisms, coccoid and bacillary, with some short filaments staining irregularly. Cultures were positive as before.

Blood.—A blood count was made, November 12. There were 5,100,000 red cells, with a white count of 28,100. Of these, 66 per cent were polymorphonuclears; 28 per cent small mononuclears; 3 per cent large mononuclears and 3 per cent transitionals. The erythrocytes appeared normal; one normoblast was seen. Blood culture was not made.

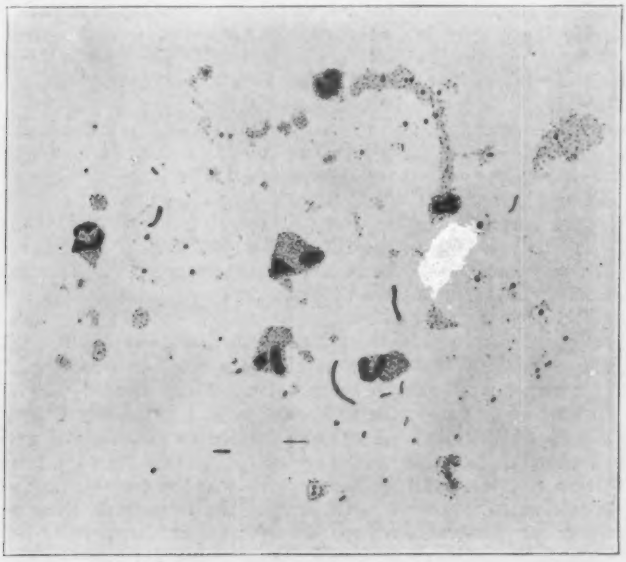

Fig. 1.—Smear of spinal fluid, uncentrifugalized. Gram's stain, showing coccoid, short bacillary and filamentous forms of the organisms. Drawing made from photograph.

MORPHOLOGY

The organism found in the spinal fluid and later in cultures offers many interesting phases when studied with various stains. Repeated attempts to stain it by the Ziehl-Neelson method resulted each time in a clean slide, with nothing to suggest the cloudy appearance of the spinal fluid. Smears stained with Loeffler's methylene blue showed a few organisms, varying from long bacilli to very short bacillary or coccoid forms. The body of these organisms stained irregularly, suggesting very slightly the staining properties of a fungous filament. Gram's method of staining, with dilute fuchsin as a counterstain, gave very satisfactory material for study. Many organisms, all gram-negative, deeply red stained, with slightly hazy outline and often with the irregularly stained body before noted, varied in size and shape from large bacilli with slightly rounded ends to short coccoid forms. Occasionally, a clubbed form was found. Measurements of the organism made from these slides varied from 0.75 to 11.7 microns in length, and from 0.19 to 0.8 microns in width, with an average width of 0.6 microns (fig. 1).

Smears from cultures showed most startling and puzzling forms. Forty-eight or seventy-two hour vigorous blood broth cultures gave deeply red stained masses of what resembled slightly branched filaments or mycelium, with swollen buds of varying size (fig. 2). Large coccoid forms were frequently seen, and often what looked like chains of budding yeast cells. Occasionally a plump bacillus was encountered. From older cultures smears presented a somewhat feathery, reticulated mass, less deeply stained with fuchsin, with enmeshed or adherent deeply stained plump, raggedly outlined coccoid forms.

Loeffler's methylene blue presented a greatly contrasting picture. It was found necessary to stain as short a time as possible to prevent the organisms from washing from the slide. When the smears were fixed with heat, the organisms stained faintly and irregularly, often with a deep bipolar stain, especially in old cultures,

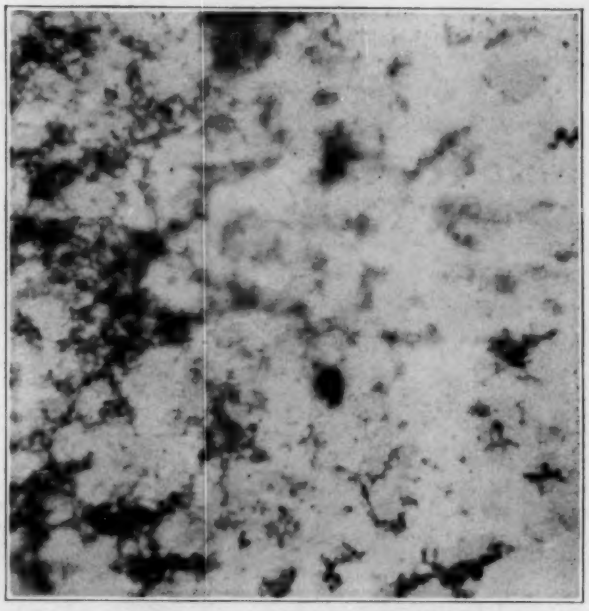

Fig. 2.—Three day old broth culture made at necropsy from blood of inoculated rabbit. Gram's stain.

in which short, somewhat barrel-shaped forms with polar bodies were frequently seen as well as long chaining forms (figs. 3 and 4). Coccoid forms were much less frequently found. Sometimes slender pointed bacilli were seen. Delicately stained feathery tracery clung to many of these organisms, but the background of the smear was clear and colorless. Smears fixed with alcohol instead of heat, or heat followed by alcohol (95 per cent), and stained with methylene blue showed a very faintly stained blue field containing colorless vacuoles of varying sizes, usually oblong (fig. 5). Many of these were empty, some contained faintly stained organisms, varying in shape from bacilli, single or in pairs, though not adjacent, to ovoid and coccoid forms. One distinctly clubbed bacillus was seen. Polar bodies did not show by this method, nor was there any structural differentiation.

Capsules stains were tried wholly without success, all material washing from the slide. The organism took aniline gentian violet stain deeply, presenting the same form as when decolorized and stained with fuchsin. So-called "choro-iodide"

of zinc[3] gave a uniformly yellow stain, the material presenting a coarsely granular appearance. It was found that acids washed all material from the slide. Aqueous stains had to be used with great care, and even then many organisms were washed

Fig. 3.—Two fields from 7 day old broth culture, third generation, from spinal fluid. Fixed in flame; stained with Loeffler's methylene blue. Width of bacilli in this group varied from 0.41 to 1.56 microns.

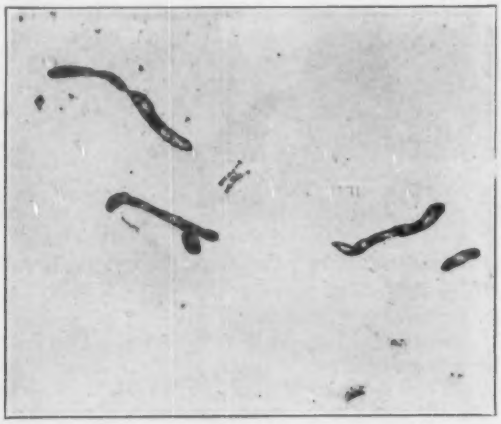

Fig. 4.—Isolated fields from 3 day old broth culture of heart's blood of a guinea-pig. Fixed in flame; stained with Loeffler's methylene blue. Width of bacilli in this group varied from 0.58 to 1.1 microns.

off. Treated with equal parts of aniline oil and 95 per cent alcohol, without staining, the material presented glistening forms similar to those seen with aniline gentian violet and with Gram's stain.

3. Stevens, W. C.: Plant Anatomy, Philadelphia, P. Blakiston's Son & Co., 1924.

The explanation of these contradictions seems to be that *Actinobacillus* produces an exudate which forms an immediate capsule-like covering for the organism, often several bacilli being held together by this means. It would seem that some portion of this coating goes into solution in the medium, making the delicately stained blue matrix in the alcohol-fixed methylene blue slides, and the few feathery wisps that were not washed from the methylene blue slides fixed by heat only. Acids evidently dissolve this exudate completely, and water dissolves it easily; alcohol dehydrates it and fixes it to the slide. Aniline oil dehydrates so energetically that it causes a contraction of the material around the organisms, the matrix wrapping itself around the bacilli and tending to produce spherical and filamentous masses. The organisms thus ensnared make the invisible framework for the otherwise wholly inexplicable, seemingly filamentous

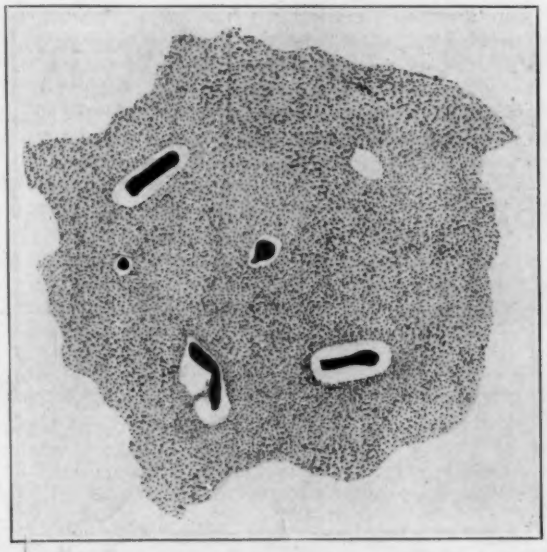

Fig. 5.—Isolated fields, co mposite drawing, 10 day old broth culture of blood from inoculated rabbit. Fixed in flame, then alcohol; stained with Loeffler's methylene blue. Average width of these organisms was 0.9 microns.

branches and buds. When bacillary forms are enmeshed, apparent filaments result, while coccoid forms appear as the yeast-like or budding branches. There was never true branching.

After repeated transplants over a period of several months, these bizarre structures became strikingly fewer, smears for the most part presenting coccoid and slender pointed bacillary forms against a pale red background.

CULTURAL CHARACTERISTICS

The spinal fluid was cultured on Loeffler's blood serum and on beef extract broth (plus 0.5 phenolsulphonphthalein) boiled and rapidly chilled, with a drop of human blood added, but not shaken through the medium. Inoculation was made by pipet with about 1 mg. of spinal fluid. In subcultures, the usual laboratory mediums were used, aerobically and anaerobically, with and without blood or ascitic fluid. The freshly isolated organism was anaerobic, becoming facultatively aerobic with repeated transplants. A noticeable feature in our

work is that at no time did growth occur without enrichment with body fluid, and heavier growth occurred in the presence of blood than with ascitic fluid.

Cultural results were largely negative. Of the solid mediums no visible growth was obtained with Loeffler's blood serum, plain and dextrose blood agar slants, blood agar shake tubes, Russell's medium enriched with blood, or potato. Anaerobic blood agar plates and lactose agar plates enriched with ascitic fluid, when heavily seeded with pipet, gave confluent growth.

Discrete colonies were obtained only on anaerobic ascitic fluid lactose agar slants. After forty-eight hours, glistening pin point colonies appeared, visible with a hand lens. These slowly increased in number during several days, but did not become confluent nor increase in size.

Of the liquid mediums, most satisfactory results were obtained with beef extract broth, as described in the foregoing. No growth was obtained with litmus milk. No indol was formed in Dunham's peptone water. No gas or noticeable acid were formed. Titrations made on the dextrose and lactose cultures showed a slight increase in acidity in the dextrose medium.

A peculiarity of this organism, which is worthy of note, was the fluffy ball of growth in the bottom of the tube when the spinal fluid itself was incubated, and when the blood from inoculated animals was cultured in broth. When touched with a loop, a delicate festoon trailed out from the main growth, but remained adherent to it. This characteristic did not appear in subcultures. The same characteristic was true to a less extent of the urine from the inoculated rabbit when it was seeded into broth. At necropsy, this urine was obtained with a sterile pipet through the seared bladder wall.

Viability.—Growth did not occur at room temperature. Urine from an inoculated rabbit was seeded into plain broth and incubated for two weeks, at the end of which time it showed no growth. It was then transferred into blood broth, when typical growth occurred. Blood from the rabbit was kept at room temperature for two weeks during cold weather, and gave typical growth when seeded into broth at the end of that time. Spinal fluid was kept in the incubator for three months, during which time it dried down to a hard, yellowish lump in the bottom of the conical tube. Plain broth was added, and after forty-eight hours this was transferred by pipet to a blood broth tube, when characteristic growth appeared, which gave typical organisms when stained. This resistance to drying is suggestive when compared with Wright's[4] work on actinomycosis.

ANIMAL INOCULATIONS

On November 16, a rabbit was inoculated intravenously with 0.4 mg. of spinal fluid, which had been kept in the incubator for five days, from November 11. The only clinical symptom which developed was reduction in the quantity of urine excreted. This urine was very cloudy and thick. The rabbit was killed at the end of three weeks. Necropsy revealed no granules or pus flakes, nor any abnormal findings with the exception of one or two slightly enlarged mesenteric lymph nodes, and some bronchopneumonia. The heart's blood and the urine yielded the same organism in pure culture as that present in the spinal fluid. No lesions could be demonstrated in sections of the various organs except the bronchopneumonia.

On November 15, a guinea-pig was inoculated intraperitoneally with broth cultures of the spinal fluid. This pig died, December 13. Necropsy revealed the peritoneal, pleural, and pericardial surfaces somewhat hemorrhagic and quite moist, with a noticeable increase in the amount of pericardial fluid. The mesenteric lymph nodes were slightly enlarged. The lungs showed scattered areas of bronchopneumonia. There was no abscess at the site of inoculation, and no granules or pus flakes were found. Blood from the heart yielded a pure culture of the organism. No lesions could be demonstrated in the tissue sections except the bronchopneumonia.

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4. Wright, J. H.: Pub. Mass. Gen. Hosp. Boston **1,** 1905.

REVIEW OF THE LITERATURE

Believing that in this case we have found a true human infection with *Actinobacillus,* and being able to find reference to only one other such case, we have attempted to review reports of related infections. Of the many cases reported of human infection due to *Actinomyces,* to *Cohnistreptothrix,* to *Discomyces,* to *Streptothrix,* and to other members of this family, the lesions are variously located. The earlier accounts describe generalized actinomycosis, with probable dental or pulmonary origin.[5] The literature has become so extensive that we have been forced to limit our studies to those reports dealing with lesions of the nervous system. These include brain abscesses,[6] sinus thrombosis,[7] and meningitis.[8] Of the thirty-eight cases of meningitis to which we have found reference as caused by organisms of this group, a large majority were secondary infections, many of them following otitis. With but one exception, the report of Pinoy and Ravaut,[1] the causative organism in these cases seems to be named, and in most cases described, as one of those which at present is included with the actinomyces. Often the laboratory report is so brief as to make any linking of the reported organism with a recognized species impossible. Though Pinoy and Ravaut report little first hand bacteriologic work on the organism, they unhesitatingly name it *Actinobacillus* of Lignières and Spitz, and state that Lignières confirms them in their diagnosis. Williams' report of the organism found in Elmendorf's and Neal's case is very suggestive of *Actinobacillus,* except for the fact that she reports branching on lactose agar. Bell (footnote 2, second reference), in a comprehensive review of *Streptothrix* infections, suggests that certain cases reported by Rutelli, Sindoni, and Kharina-Marinucci,

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5. Israel, J.: Virchows Arch. f. path. Anat. **74,** 1878; ibid. **78,** 1879; ibid. **126,** 1891. Flügge, C.: Micro-Organisms with Special Reference to the Etiology of the Infective Diseases, tr. by Cheyne, London, The New Sydenham Society, p. 140. Koch, R., and Flügge, C.: Ztschr. f. Hyg. u. Infectionskrankh., 1901, p. 345. Klinger, R.: Untersuchungen über menschliche Aktinomykose, Centralbl. f. Bakteriol. **62:**191, 1912.

6. Steele, A. E.: A Streptothrix Organism from a Brain Abscess, J. M. Res. **44:**305, 1924. Eppinger: Beitr. z. path. Anat. u. z. allg. Path. **9,** 1890. Colebrook, L.: Mycelial and other Micro-Organisms Associated with Human Actinomycosis, Brit. J. Exper. Path. **1,** 1920.

7. Horn, H.: Ann. Otol. **22:**989, 1913.

8. Neuman, R., and Rabinowitsch-Kemper, L.: Ztschr. f. klin. Med. **94:**215, 1922. Sternberg, Carl: Verhandl. d. deutsch. path. Gesellsch., 1910, p. 215. Von Eicken and Huntemüller: Deutsche med. Wchnschr. **39** and **40,** 1919. Naunyn: Beitr. z. path. Anat. u. z. allg. Path., 1891. Rutelli: Pediatria **23:**713, 1915. Sindoni: Pediatria **24:**530, 1916. Kharina-Marinucci: Pediatria **26:**11, 1918. Fonzo: Pediatria **30:**1045, 1922. Bernhardt: Klin. Wchnschr., no. 25, 1914. Beevor, C. E., and Farquhar Buzzard, E.: Tr. Path. Soc. London **54,** 1903. Elmendorf, T. E., and Neal, J. B.: Streptothrix Meningitis, Arch. Pediat. **42:**260, 1925.

which these authors attribute to *Streptothrix*, may be *Actinobacillus* of Brumpt.[9] Our own impression from their original reports leaves us doubtful that they were working on *Actinobacillus*. There is an absence of any description of the "bizarre forms," and especially of the "coccobacilli" found in *Actinobacillus* cultures. The plates of Rutelli are somewhat suggestive of our organism, but Sindoni, working with an "identical" organism, states that "the extremities may be forked."

Pinoy and Ravaut [1] report the first case of meningitis from which an organism identified as *Actinobacillus* of Lignières and Spitz was recovered. The patient reported by them was a youth, aged 16, who in January, 1909, had otitis media and mastoiditis, from which he made an uneventful recovery after operation. In February of that year he developed meningitis, showing marked torpor with absolute indifference to all that happened around him, failing to respond to questions asked. He had violent headache, photophobia, vomiting and constipation, stiffness of the neck, some opisthotonus and a positive Kernig's sign. Lumbar puncture yielded a clear spinal fluid under pressure, showing definite reactions of meningitis. Smear showed no organisms, but culture after three days incubation showed small colonies of a gram-negative coccobacillus. "By its morphology and cultural characteristics, the organism isolated from the spinal fluid" of this patient was identified as "*Actinobacillus* similar to that which Lignières and Spitz" had described. They state that: "This is the first time that *Actinobacillus* has been recovered from the disease in a human being." The patient recovered and returned to Argentina, where he had formerly lived.

Actinobacillus has been studied by a relatively small number of workers. Primarily, it is a disease of cattle, presenting a picture closely identified with that of actinomycosis. From this, its clinical picture differs in that *Actinobacillus* has a predilection for attacking the lymph glands, in addition to causing abscesses of the jaw. Bacteriologically, it differs from actinomycosis in that its causative agent is a gram-negative bacillus, and never branches. In 1900 and 1901, the disease became epizootic in Argentina, at which time Lignières and Spitz [10] made a thorough study of its cause, and their work is the authoritative account on which all later study is based. In 1902, Nocard [11] apparently obtained identical results, and reported two bovine cases in France. Griffeth,[12] working in England, concluded that "*Actinobacillus* is wide-

9. Brumpt. E.: Précis de Parasitologie, Paris, 1913, p. 963.

10. Lignières, J., and Spitz, G.: Contribution à l'étude, à la classification et a nomenclature des affections connues sons le nom d'actinomycose, Centralbl. f. Bakteriol. **35**:436 and 452, 1904. English review in J. Comp. Med. & Vet. Arch. **103** (May) 1903.

11. Nocard, E.: Bull. Soc. Cen. Med. Vet., 1902, p. 695.

12. Griffeth, F.: J. Hyg. **15**:195, 1915-1917.

spread in the world, and forms a considerable proportion of the cases of disease in oxen known under the name of actinomycosis." This conclusion is based on the examination of fifty tongues and lymph glands obtained from a slaughter house in London, forty-six frozen specimens from Argentina, two from North America, and two from Siberia. The dissimilarities in his cultural results from those of Lignières may be explained by the different mediums used. Griffeth and Higgins,[13] who worked on four cases of bovine actinobacillosis in Canada, were both working with the disease in its mild sporadic form, and the results of animal inoculations were not as marked in virulence as those of Lignières. Chaussé, who states positively that, in the cases of bovine actinobacillosis which he studied, he found gram-positive clubbed organisms surrounding gram-negative bacilli in the center of a granule, is in direct contradiction to all other writers on this disease. His report suggests mixed infection, and a comparison with Colebrook's (footnote 6, third reference) work on *Bacillus actinomycetum-comitans,* in which a gram-negative bacillus was found as a constant accompaniment to the gram-positive filaments and clubs of his actinomycotic granules. Wright,[4] in his work on twelve cases of bovine actinomycosis, isolated from one case a gram-negative organism which clouded broth. This would suggest the possibility of *Actinobacillus.* Koch and Flügge (footnote 5, fifth reference) mention a case which, though briefly described, suggests certain similarity with Lignières' organism.

The method by which actinobacillosis spreads, even among cattle, has never been determined, though thorough studies have been made with this in view.[10] In the case here reported, we have been unable to locate the source of the infection. The patient had been put on cow's milk feedings just about the time of the onset of the disease, and had also been given condensed milk canned by a reliable firm. Examination by a veterinarian of the cows from which the fresh milk was obtained showed no lesions suggesting this infection. The first can of condensed milk used in feeding had been entirely consumed. A second can had been opened for use just prior to the transferring of the patient to the hospital, and after the symptoms were well developed. Cultures from this milk did not show any growth which could be identified as this organism. The company marketing this condensed milk said that they do not import milk from the South American countries, but that some of their supply comes from Canada. We do not feel that we have any evidence suggesting the possible source of this infection.

"Iodide of potassium, it is said, is an excellent specific, and under its prolonged use the lesions gradually disappear, except when the

13. Higgins, C. H.: Actinobacillosis, Ottawa, Canada, Department of Agriculture, 1904.

bones are affected. From 10 to 12 Gm. are given daily (for cattle), or 1 mg. of a 10 per cent solution of 1 mg. of compound solution of iodine are injected into the thickness of the tumor" (footnote 10, second reference). We did not use potassium iodide with this patient.

SUMMARY

Although primarily an infection of cattle, organisms of *Actinomycetaceae* do cause human infection.

Only one other case of human infection by *Actinobacillus* has been reported.

The organism in the case here reported seems to be *Actinobacillus* (Brumpt) of Lignières and Spitz.

The source of the infection has not been determined in this case.

HODGKIN'S DISEASE, PRIMARY IN THE THYMUS GLAND

REPORT OF A CASE IN AN INFANT*

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AND

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Lesions of the thymus gland in young infants are among the less common conditions encountered, either clinically or at necropsy. Acute inflammatory lesions are rare. Hyperplasia accompanying status thymicolymphaticus is the thymic lesion seen most frequently. Other causes of enlargement are neoplasms, Hodgkin's disease and leukemia.

The following case is reported in detail because some of its features differ from any hitherto recorded:

REPORT OF CASE

A white, female infant, aged 4½ months, born in this country, of Irish parents, was admitted to the Babies' Hospital, September 29, and died, Oct. 1, 1924. Her father and mother were alive and well. The patient was the seventh child. The other six were in good health. No miscarriages or stillbirths had occurred, nor was any history of familial disease elicited from the parents.

The infant was born at term after a normal labor. The weight at birth had not been determined. She had been nursed "whenever she cried," and in addition had been given small amounts of diluted milk. The administration of orange juice in varying amounts had been begun one month before admission to the hospital.

The mother stated that the infant had thriven for the first three months of life, and then a "roughness of the palms of the hands" had been noted. This gradually became more marked until the palms and soles "felt like sandpaper." Soon afterward, fine "red spots" made their appearance. Two weeks later "spots" were seen on the abdomen, behind both ears, and on the back of the head, increasing in extent and intensity. After three weeks spots appeared on the back. The mother further stated that there was a variability in the distribution and intensity of the eruption from time to time, with a definite tendency for it to involve more of the skin surface. Apparently the eruption increased in extent from the onset, coming out in distinct crop-like exacerbations. At no time had there been any bleeding or oozing from the skin, nose or anus, although oozing of blood from the gums occurred after digital cleansing of the mouth. Blood was never recognized as such in the stools or urine. Three weeks before admission, the character of the stools changed from normal to what was described as "green and slimy"; there were from three to five in twenty-four hours.

Fever was present for four days prior to admission, fluctuating around 101 F. When the hyperpyrexia began, the mother noticed that the infant cried whenever she was disturbed or handled.

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* Received for publication, June 28, 1926.

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Physical Examination.—A well nourished infant, slightly dyspneic, who appeared to be acutely but not critically ill, was seen. The systemic disturbance seemed relatively slight in comparison with the extent of the integumentary involvement. The weight was 13 pounds 10 ounces (6 Kg.), and the height 26¾ inches. The head measured 16¼ inches, the chest 15½ inches, and the abdomen 15¾ inches. The temperature was 102.4 F. on admission. The lips and conjunctivae were pale.

Fig. 1.—Petechial rash on skin of abdomen, chest, neck and palms.

An extremely profuse, purplish-red petechial eruption covered the entire back from the occipital region to the coccyx (figs. 1 and 2). It was distributed with equal intensity over the abdomen, over both flanks and over the chest as high as the fourth rib. From the fourth rib to the suprasternal notch, the petechial spots were smaller, more uniform in size and widely scattered. In the groin, between the gluteal folds, in the axillae, in the posterior triangle of the neck, and over the palms of the hands and the soles of the feet, where the skin is subjected to more frequent friction and hence to more trauma, the eruption was of a more confluent hemorrhagic character, due to the coalescence of the individual petechiae.

Over the last named areas the skin was purple, almost black. The extremities were remarkably free from eruption as compared with the trunk, but there were clusters of hemorrhages in the popliteal space, over the dorsum of the feet, between the toes and on the soles. There was also a group in each cubital fossa, and these ran upward to join the spots in the heavily involved axillae. There were scattered petechiae over the dorsum of the hands and a profuse eruption

Fig. 2.—Petechial rash on skin of neck, shoulders and back.

on the palms and over all the fingers. The face was entirely free. Along the hair line above the eyes and ears, over both ears, on the scalp over the occipital region, and over the upper posterior cervical region were many petechiae. Over the trunk, where the eruption was most marked, a fine, branny desquamation was present. On passing the fingers lightly over this area, a sensation of roughness was experienced, not unlike that of fine sandpaper.

The mucous membrane of the mouth was pale, with petechiae on the gums, the border of the tongue, the inner surface of the cheeks, and the left anterior faucial pillar. Both tonsils were slightly enlarged. There were no teeth. No petechiae were noted on the sclerae. The pupils were equal and reacted normally to light. The head was normal in contour, and without craniotabes. The anterior fontanel measured 3 cm. None of the superficial lymph nodes were enlarged.

The chest was normal in shape. There were no adventitious signs in the lungs. The heart was normal in size and position; no murmurs were noted.

The abdomen was soft and full. The liver was enlarged, with a readily palpable, smooth, lower border at the level of the umbilicus. The spleen was also large, with the lower pole at the level of the umbilicus. The lower extremities were slightly edematous and tender on passive motion.

Laboratory Examination.—The urine contained a faint trace of albumin, an occasional granular and hyaline cast, and some leukocytes, singly and in clumps. No red blood cells were found, and the guaiac test for blood was negative. Blood culture showed no growth after eighty-four hours. The Pirquet skin test was negative. A throat culture was negative for diphtheria bacilli. A vaginal smear showed neither gonococci nor pus cells.

The red blood cells numbered 2,800,000, and the hemoglobin content (Dare) was 55 per cent. There were 12,500 white blood cells with 64 per cent polymorphonuclear leukocytes, 33 per cent lymphocytes, 2 per cent monocytes, and 1 per cent myeloblasts. For every hundred white blood cells, 16 nucleated red cells were counted. The blood platelets numbered only 88,000. The coagulation time was two and one-half minutes.

A lumbar puncture showed crystal clear cerebrospinal fluid under normal pressure, containing four cells per cubic millimeter. Benedict's test was positive for sugar. The test for globulin was negative.

Progress.—During the two day period of hospital observation, the temperature fluctuated between 99.6 and 102.2 F., with an antemortem fall to 97 F. The stools were thin, with a small amount of fecal matter, and were passed not oftener than four times in twenty-four hours. The infant was nursed by her mother, and the only recorded amount taken from the breast at a single nursing was 200 cc. Suddenly and unexpectedly, on the third day after admission, the child died in the arms of the nurse who was carrying her to the operating room for a transfusion. A roentgenogram of the chest was not made.

Necropsy.—The necropsy disclosed the following: The body was that of a well formed and extremely well nourished white female infant. Over the skin on the chest, abdomen and back there were innumerable petechial hemorrhages, so that the effect was of a bright scarlet skin with dark blue dots the size of pinpoints. Petechiae were also present over the scalp and the posterior surface of the neck, and there were a few on the lower lip and on the lower gums. The nose was quite free. There were many petechiae over the palms and soles, which were slightly desquamating. The feet, legs and hands were distinctly swollen and pitted on pressure. Examination of the brain was not permitted.

Both lungs were emphysematous throughout. No nodules were present. The bronchial lymph nodes were dark red and not enlarged; they showed no evidence of tuberculosis.

The thymus weighed 28 Gm. It was firm, solid, surrounded by its capsule, and quite free, not having invaded any surrounding structure. The color was red, streaked with yellow. The gland was composed of the usual right and left lobes and a central lobe. The right lobe was 4 cm. long, the left 5 cm., and the central lobe 3 cm. in length. The entire width was 4.5 cm. The mass was roughly rectangular in shape, and the central lobe extended above the level of the other two for a distance of 1 cm., while at the lower border the left lobe was below the level of the right (fig. 3). The posterior surface was concave where it received the heart. On section, the lobulations were very coarse; the septums were much thicker than is usual in the thymus, and the cut surface was mottled red and yellow.

The spleen weighed 106 Gm.; it measured 8 by 5 cm., and was red, with small, dark areas of hemorrhage. The capsule was smooth and the substance extremely friable, so that it broke under the slightest pressure. No nodules were palpable or visible, and no tubercles were present.

The liver weighed 330 Gm.; it was mottled red and yellow due to congestion and fatty change. No nodules were present, and the capsule was not thickened.

The stomach and duodenum were normal. Throughout the small intestine, all the solitary follicles and Peyer's patches were congested and swollen. The mucosa of the ileum and colon was congested and covered with mucus. Just outside of the cecum lay a group of lymph nodes, the largest 0.5 cm. in diameter. Two contained pinhead sized, yellow, softened areas, like small abscesses. All the mesenteric nodes were slightly swollen, and contained small hemorrhages; the mesentery was edematous. The retroperitoneal nodes were small but bright red in color. None were tuberculous.

Fig. 3.—Thymus gland. Note coarse lobulation.

The kidneys weighed 40 Gm., and were firm and pink. The capsule was free, the markings distinct and no nodules were present.

The ureters, bladder, peritoneum, uterus and ovaries showed nothing abnormal.

The anatomic diagnosis was: thymoma; petechial hemorrhages into the skin; subcutaneous edema; pulmonary emphysema; acute splenic tumor; nutmeg liver, and acute catarrhal enterocolitis.

A culture made from the heart's blood grew *Staphylococcus aureus.*

Microscopic Examination.—Section of the thymus did not resemble the normal thymus gland. Hassall's corpuscles were not present at first glance, nor was the normal lobular structure seen. Instead, there were bands of connective tissue with many fibroblasts running irregularly through the section, outlining large lobules, some of which were subdivided by narrower connective tissue strands into smaller compartments, irregular in size and shape. The periphery of many lobules was densely packed with small, mononuclear cells like the lymphocytes of the normal thymus gland. The central portion, less dense, contained cells of several

kinds, the most numerous being comparatively large, round or ovoid, with vesicular nuclei eccentrically placed, and an acidophilic cytoplasm. From two to four nuclei were not uncommon. Such cells were distinct close to a blood vessel and sometimes surrounded a small artery, widening its wall. The more delicate, paler staining and irregularly shaped reticulum cells were plainly distinguishable, a few with hyperchromatic nuclei. Many polymorphonuclear leukocytes and a few eosinophils made the picture more mixed (fig. 4). Hemorrhage had occurred in the central part of some lobules, and large mononuclear cells had phagocyted brown pigment granules. Areas of focal necrosis were also present. It was possible to find one or two small Hassall's corpuscles with their central keratinized cells and a few concentric rings. These few remaining bodies gave evidence of the diffuseness and severity of the inflammatory process, which had practically obliterated the normal architecture of the thymus gland. The absence of younger Hassall's bodies may be looked on as contributing evidence that the process was

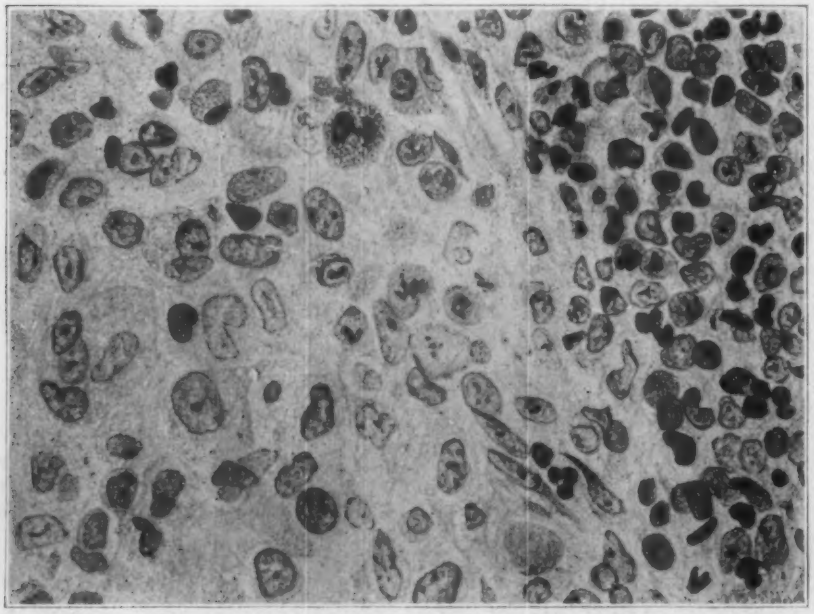

Fig. 4.—Thymus gland showing the great variety of cells within the lobules.

not neoplasmic in type. An area of thick fibrous connective tissue with neighboring cellular replacement indicated a much older stage of the granulomatous lesion.

The capsule of the spleen was normal in thickness. It was impossible to outline any malpighian bodies, but there were a few small irregular collections of lymphoid cells around small arteries. The connective tissue septums were normal. The sinuses were all dilated and filled with red blood cells. Between the sinuses were many large mononuclear cells and a few eosinophils. There were small areas of hemorrhage, practically no pigment and no necrosis.

The capsule of the liver was normal. Cells in the periphery of the lobules contained a large fat drop. The sinusoids were empty. In the interlobular connective tissue were groups of cells made up of large monocytes, eosinophils and polymorphonuclear leukocytes.

The capsule of a bronchial lymph node was intact. The sinuses contained numerous endothelial cells, as did the central portion of the lymph nodules, leaving

only a narrow rim of lymphocytes. An occasional giant cell was present. Neither hemorrhage nor necrosis had occurred.

An abdominal lymph node, adjacent to the pancreas, showed a more advanced lesion of endothelial hyperplasia and necrosis. Its subscapular sinus was packed with proliferating endothelial cells, which also filled the peripheral portion of the adjacent nodules. In other nodules the entire center was made of a mass, or sheet, of endothelia, outlined by small lymphocytes. Cells containing three or four nuclei were present. Phagocytosis was going on by large cells which had ingested fragments of nuclei, whole lymphocytes or whole red blood cells. The blood vessels contained an occasional eosinophil. Areas of necrosis surrounded by poly-

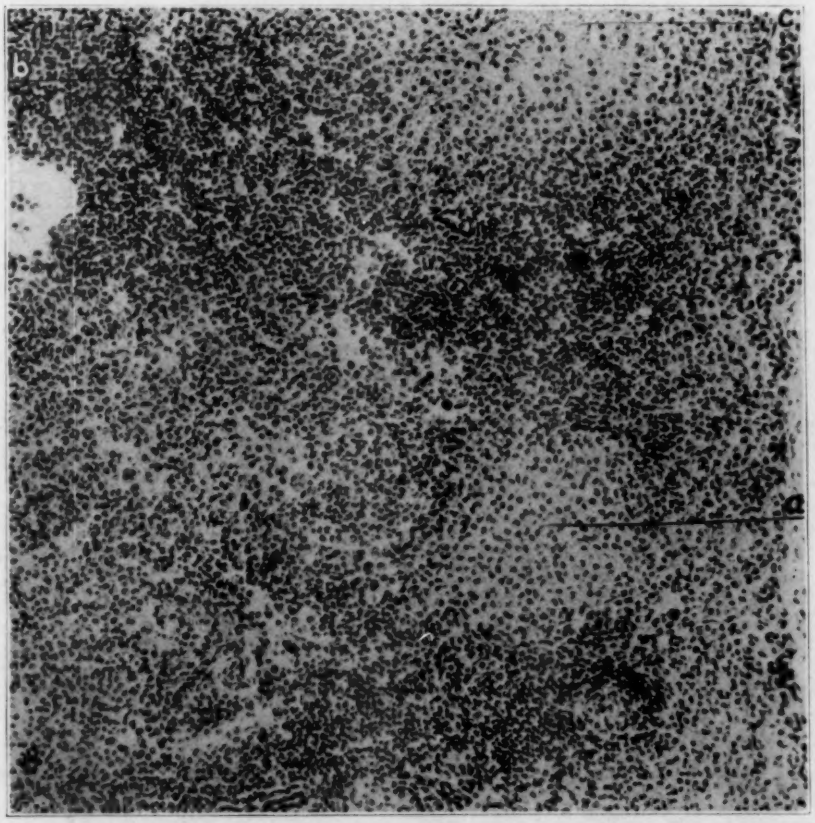

Fig. 5.—Lymph node, showing large cells in center of nodule, *a;* lymphoid cells at periphery, *b*; focal necrosis, *c*; × 160.

morphonuclear leukocytes were seen, especially in the nodules containing many endothelial cells (fig. 5). Small hemorrhages had occurred within such nodules, evidently preceding the necrosis. There was no fibrosis. The normal outline of the nodules and cords was practically obliterated. Tubercles were not present, and tubercle bacilli were not found.

The lining cells of the colon were entirely gone. The glands were intact and contained many beaker cells. The interglandular tissue was unchanged, but a small lymph nodule in the submucosa contained many endothelial cells. An

adjacent mesenteric lymph node showed much endothelial hyperplasia in its subcapsular sinus and in its nodules, with multinucleated cells which were not larger than the mononuclears.

The epithelium of the convoluted tubules of the kidneys was in good condition. The glomeruli varied somewhat in size, and were for the most part normal. The collecting tubules were normal. In the cortex there were a few collections of smaller and larger mononuclear and multinucleated cells between groups of tubules. These collections were small and present especially beneath the capsule.

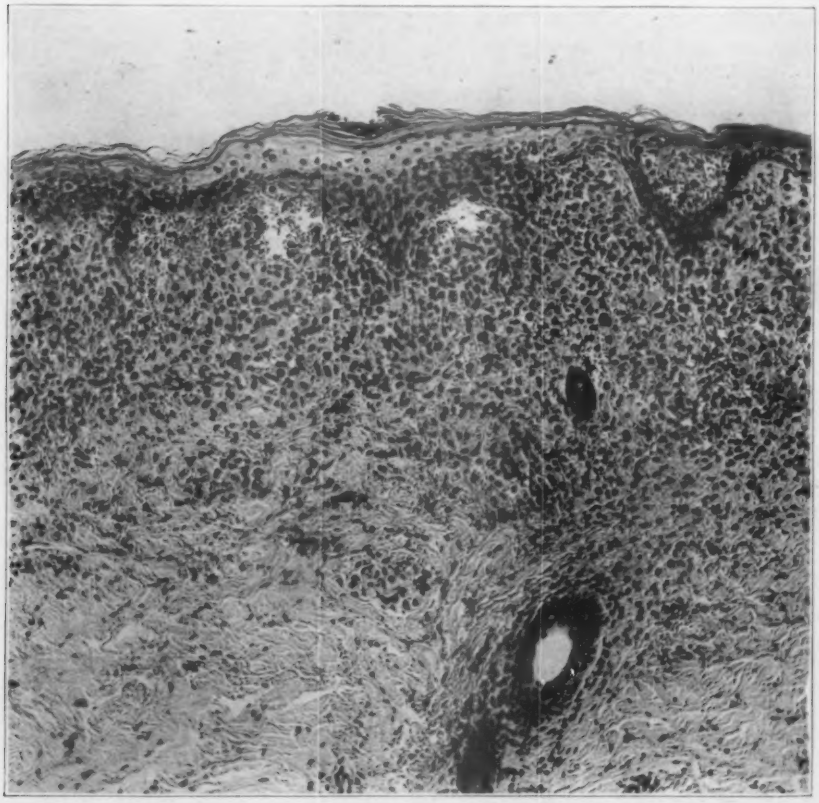

Fig. 6.—Skin showing variety of cellular infiltration around small hemorrhages in corium (× 160).

The horny layer of the epidermis was irregular in thickness and desquamating. The whole epidermis was of normal thickness. So was the corium, in which hair follicles, sebaceous and sweat glands were normal. In the papillae of the corium there were numerous small hemorrhages just beneath the epidermis. These hemorrhages were surrounded by masses of cells, some large and mononuclear, a few with many nuclei, lymphocytes, a few polymorphonuclear leukocytes, and an occasional eosinophil. Some of the hemorrhages, with their surrounding infiltration, extended quite deeply into the corium, and others had pushed aside the cells of the rete (fig. 6).

The entire lesion was that of an infectious granuloma, affecting the thymus gland, the skin, many abdominal and some thoracic lymph nodes, the spleen and,

to a less extent, the liver and kidneys. A diagnosis of Hodgkin's disease primary in the thymus gland was made, and the gross diagnosis of thymoma (or lymphosarcomatous neoplasm) was not confirmed.

COMMENT

The illness of this infant began with petechial hemorrhages into the skin. Six weeks later the blood picture was that of an anemia with low hemoglobin and low red cell count, a slight polymorphonuclear leukocytosis and a diminution in the number of platelets. The low platelet count of 88,000 was striking, and the diagnosis of thrombocytopenic purpura was considered. The petechial skin hemorrhages made the outstanding symptom, but the infant was under observation only three days. Evidently she had not been ill sufficiently before that to be brought for observation, although fever had been present for four days. No superficial lymph nodes were enlarged, which is an unusual but not a unique circumstance. The striking feature was the combination of skin hemorrhages, thymic tumor, fever and rapid death, six weeks after the first symptoms were noted.

The necropsy observations were a surprise, since cyanosis had been absent, and the mild dyspnea had not suggested a possible mediastinal tumor. Microscopically, the thymic tumor showed the picture of an infectious granuloma, with the mixture of cell types (multinucleated giant cells, large mononuclear cells, lymphocytes and eosinophils) and the degeneration areas of such a lesion. There was also the old fibrous area, indicative of the final stage of Hodgkin's granuloma. The lymph nodes showed an earlier stage of the process, without fibrosis, and in the spleen, liver and kidneys the lesion had only just begun. The skin lesions, on the other hand, were well developed and characteristic.

The case must be considered in relation to the following thymic lesions: Neoplasms, including carcinoma, sarcoma and lymphosarcoma, Hodgkin's granuloma, and lymphatic leukemia.

Lymphatic leukemia in young infants involves the thymus gland but rarely, and was ruled out by the blood picture in this patient.

REVIEW OF THE LITERATURE

Rocaz[1] has reported a case of leukemia in a boy, aged 4 years, whose thymus weighed 200 Gm.

Of the epithelial variety of thymic neoplasms, few have been recorded (seven, according to Foot;[2] nineteen, according to Brannan;[3] one by Foot and Harrington[4] in a child, aged 2 years). With the size

1. Rocaz: Rev. mens. d. mal. de l'enf. **20**:120, 1902.
2. Foot, N. C.: Am. J. Path. **2**:33, 1926.
3. Brannan, D.: Carcinoma of the Thymus and Occlusion of the Superior Vena Cava, Arch. Path. **1**:569 (April) 1926.
4. Foot, N. C., and Harrington, H.: Malignant Epithelial Thymoma, Am. J. Dis. Child. **26**:164 (Aug.) 1923.

of this tumor, its gross and microscopic picture of malignancy, the case referred to in this paper has nothing in common.

Lymphosarcomas may originate in the thymus, but they invade the surrounding organs and lymph nodes, and may involve distant organs. Such cases have been described in children by Gangee [5] and by Arnheim.[6]

One variety of lymphosarcoma of the thymus gives the microscopic picture of granuloma malignum (Ewing [7]), but the case here described differs from that group in the absence of the polyhedral type of proliferating thymus reticulum cell and of any attempt at the formation of Hassall bodies. It represents a systemic granulomatosis of the Hodgkin's type originating in the thymus gland, the lesion being inflammatory and not neoplasmic. The mass was limited to the thymus, within its capsule, and did not spread to neighboring lymph nodes, lung or pericardium. The gross appearance was that of a greatly enlarged thymus, more firm than a hyperplastic thymus ever becomes, with abnormally thick connective tissue septums, white in places, yellow and edematous in others, making irregular lobules in which the congested parenchyma was held. The rapid course may well have been due to the early age of the patient rather than to any inherent quality due to its thymic origin.

The skin symptoms which appear early in Hodgkin's disease are usually nonspecific in character, but in the case of the infant here reported they proved to be histologically specific. Grossly, they were tiny hemorrhagic nodules. Subcutaneous hemorrhages are not uncommon in Hodgkin's disease, and hemorrhage into the skin of the arms, hands and right foot were mentioned by Fischer [8] in his case of a boy, aged 5 years, who died of empyema and showed a thymic tumor, which Fischer diagnosed as malignant lymphoma, Hodgkin's disease or pseudoleukemia. Becher's [9] case, a man, aged 76, presented a widespread petechial skin lesion which, microscopically, showed the characteristic structure of granulomatosis. Karsner [10] reported a case of Hodgkin's disease in a boy, aged 17 years, who had petechial hemorrhages into the skin, attributed to the severe anemia. No platelet counts are recorded.

The blood picture in our case is consistent with that of Hodgkin's disease, which may or may not show a leukopenia. The low platelet

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5. Gangee, A.: Edinburgh M. J. **18**:797, 1873.
6. Arnheim: Berl. klin. Wchnschr. **41**:95, 1904.
7. Ewing, J.: Neoplastic Diseases, Philadelphia, J. B. Lippincott Company, 1919.
8. Fischer, O.: Inaug. Dissertation, Berlin, 1896.
9. Becher, F.: Arch. f. Dermat. u. Syph. **135**:31, 1921.
10. Karsner, H.: A Study of Cases of Hodgkin's Disease and Certain Allied Conditions, Arch. Int. Med. **6**:175 (Aug.) 1910.

count and the hemorrhagic tendency are consistent. Ziegler [11] states that blood platelets are usually diminished in Hodgkin's disease, but Bunting [12] found that they are increased.

Diarrhea is a symptom often noted. In this case the gross lesion of catarrhal enterocolitis was found, with microscopically early specific lesions in the mesenteric nodes. It is probable that the generalization of the lesion, as in tuberculosis, is due to the lack of resistance and immunity of the infant. The terminal staphylococcus infection may be regarded as purely accidental, since we know that infants succumb so frequently to this omnipresent organism, to which the immune content of their serum is lower than is the adult serum, as Tunnicliff [13] has shown. The etiology of Hodgkin's disease is still unknown.

The usual duration of Hodgkin's disease makes it, of necessity, rare in infancy. However, acute cases in adults have been known to run a fatal course in four weeks, and Lehndorf [14] says that the duration is shorter and the course more malignant in young subjects. Benjamin [15] found malignant granuloma not rare in childhood, but rarer than leukemia. That, in our experience, would make it at least uncommon. Ziegler [11] analyzed 220 cases, of which thirty-six subjects were under 10 years of age, the youngest being 5½ months old. It is his opinion that skin changes occur in one fourth of all cases, and that, occasionally, as a sign of hemorrhagic diathesis, small petechiae or larger hemorrhages may occur. Hippel [16] regarded the petechiae and larger hemorrhage in his patient, a girl, aged 7 years, as due to a hemorrhagic diathesis. According to Benjamin [15] one third of the cases of Hodgkin's disease in children are accompanied by mediastinal tumors, but true granulomatous skin lesions have not, as yet, been observed. The patient described in this paper had a skin lesion which was specific. The sandpaper-like feel of the lesions during life proved that they were more than simple hemorrhages, which are smooth.

The case of the youngest patient with Hodgkin's disease in the literature available to us is described in detail. It shows a rare combination of thymic tumor; hemorrhagic skin lesions specific in type; a blood picture of slight leukocytosis, diminished red cells and hemoglobin, with a low platelet count; early involvement of abdominal and thoracic lymph nodes, without enlargement of any superficial nodes, and death six weeks after onset of symptoms.

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11. Ziegler, Kurt: Die Hodgkinische Krankheit, Jena, 1911.
12. Bunting: Bull. Johns Hopkins Hosp. **22**:114, 369, 1911; **25**:173.
13. Tunnicliff, Ruth: J. Infect. Dis. **7**:698, 1910.
14. Lehndorf, H.: Jahrb. f. Kinderh. **67**:430, 1908.
15. Benjamin, E., in Pfaundler and Schlossman: Handbuch der Kinderkrankheiten, Leipsic, **1**, 1923.
16. Hippel, B.: München. med. Wchnschr. **57**:384, 1910.

HELIOTHERAPY AND ACTINOTHERAPY IN RELATION TO PEDIATRICS*

FREDERIC W. SCHLUTZ, M.D.

MINNEAPOLIS

I

Light therapeutics, both natural and artificial, is gradually assuming a more important place in the treatment of disease. Heliotherapy is now considered a curative agent *par excellence* for certain maladies. Michelet's old adage, "Of all the flowers, that which most needs the sun is the human flower," has been proved scientifically. Light as a curative agent has acquired its reputation largely within the last twenty years. But only within the last eighteen months has it been demonstrated how light might act on the animal organism. It appears that physiologically active substances which perfect the desired cure are produced by irradiation from inactive precursors in the foods or tissues of the animal. Drummond (1925) states that the evidence in support of this theory has gradually been forthcoming, and its unfolding constitutes one of the most remarkable series of events in modern biochemistry.

HISTORICAL

The use of light for the treatment of disease is not of as recent origin as one might think. Since the earliest times, physicians have recognized the value of sunlight in the treatment of disease. Many references to light as a therapeutic agent can be found in the works of Herodotus, Hippocrates, Celsus and Galen. Pliny stated, "Sol est remediorum maximum." The Greeks and Romans built solaria in their homes. The ancient Egyptians worshipped a sun god, and recognized the beneficial action on disease obtained by visiting sandy beaches. As early as the fourth century A.D., the Greek physician Oribasius attempted to popularize heliotherapy. Pliny the younger also advocated the use of light. In the early literature of that time, there are indications that artificial sources of light were used. The Germans, according to their religious creeds, exposed their sick infants to the sun's rays. They believed that the sun furnished Freya, queen of the heavens, with an efficient means of combating all disease.

Avicenna, recognizing the importance of heliotherapy in the earlier writings, adopted methods for the use of sunlight which served as a basis for our modern technic. Paracelsus was probably the first to call attention to the value of the climate of a mountainous district as a

* Received for publication, Sept. 4, 1926.

therapeutic aid. He recommended the district of Saint Moritz. However, considerable credit is due to Jean Jacques Rousseau, who popularized the mountainous climate in "Novelle Héloise."

It appears that the French are really responsible for the revival and continued use of the therapeutic agent, light. It is considered that the benefit derived from pilgrimages made to shrines and to certain popular physicians of the times was due largely to the exposure of the pilgrims to the sunlight during their long journeys. Roy was one of the early popular healers who claimed to cure scrofula by laying his hands on those diseased. Many times the pilgrims did not reach him, but returned home improved or cured by the journey. Probably the first report of interest on this subject was presented by Faure before the Royal Academy of Paris in 1774. He dealt with the treatment of scars, wounds and tumors by sunlight. Lapeyre (1776) was successful in treating ulcers by the sun's rays concentrated with a lens. Two years later, Faure and Lecomte presented case reports of ulcers and cancer of the lip which they cured by heliotherapy. Bertrand, in 1799, contributed evidence as to the value of light in medicine. Cauvin published a thesis, "Des bienfaits de l'insolation," in 1815. In this he advises parents concerning their scrofulous infants as follows: "Envoyez-les à la campagne, nourrisez-les le mieux que vous pourrez, mais surtout faites-les rôtir, brûler, rôtir au soleil." Diseases for which he recommends sunlight as "un moyen curatif efficace" are scrofula, rickets, scurvy, rheumatism, paralysis, muscular weakness and dropsy. He also reports a marvelous cure for a case of tuberculous rheumatism of Poncet.

Inspired by the work of Cauvin, Paulin Girard used heliotherapy for the treatment of chronic pulmonary catarrh, pulmonary tuberculosis, scurvy, dropsy, scrofula and chlorosis. He presented his paper, "Les considérations générales sur l'heureuse influence du soleil" to the Faculty of Medicine of Paris in May, 1819. At the same time Monoyer was using sunlight for suppurative arthritis, peritoneal tuberculosis and Pott's disease. About this time Grotthus formulated his law that only absorbed light rays are effective in producing chemical change. Veldes Richli, the Swiss physician, was the first to commence the treatment of disease by irradiation in a sanatorium. He also published an article entitled "Atmospheric Cure."

The discovery of ultraviolet rays by Herrschel placed heliotherapy on a scientific basis. In 1893 the important work of Finsen appeared. He called attention to the medical action of ultraviolet rays. The carbon arc lamp used by Finsen was improved by Kjeldsen. Wheatstone (1835) devised the mercury-vapor lamp as it is known now by passing an electric current through mercury-vapor. He noted that a bluish light was emitted which contained no orange or red rays. The first lamp which

attracted public attention was perfected by Cooper-Hewitt (1901). About this time Heraeus discovered that quartz crystals could be melted to make quartz receptacles. In 1904, Kromayer used water cooling attachments for the lamp. This and other suggestions of his applied to quartz lights have produced the present Kromayer lamp.

In 1899 Finsen first published his work on the use of ultraviolet rays in treating lupus. In 1902, Bernhard at Saint Moritz used sunlight to treat a suppurating wound of the abdomen. His method was based on his observation that Swiss butchers cured their meat in the sun. Bernhard soon published his technic for the treatment of surgical conditions by sunlight. Simultaneously and unaware of Bernhard's discovery, Rollier at Leysin, in 1903, began his celebrated work. Among the important investigators using light therapy about the same time were Malgat (1904), Morin (1905), Escherich (1909), Bardenheuer (1911) and von Schrötter (1912). Morin was apparently the first to treat pulmonary tuberculosis in children with light.

SOURCES OF ULTRAVIOLET RAYS

The percentage of ultraviolet radiations present in sunlight is exceedingly variable, depending on atmospheric conditions, especially the moisture present in the air. Hence it becomes necessary to obtain ultraviolet radiation artificially by one of the following means:

1. Chemical lamps which burn certain fluids in oxygen. These are seldom used, as they are difficult to operate and possess a limited field of radiation.
2. High tension disruptive elective sparks discharged between metal electrodes as, for example, tungsten.
3. Carbon arcs.
4. Mercury vapor arcs in vacuo.

The three latter sources have found application in medicine. The lamp adopted by Finsen contained a carbon arc light of high candle power. The light was concentrated by a series of quartz lenses that were water cooled. Finsen's aim was to obtain a light with the maximum of ultraviolet rays and a minimum of heat rays which cause burning. Around each light were hung four concentrating telescopes connected to an iron ring suspended from the ceiling. Although the light was cooled, it was too hot to apply directly; hence Finsen devised a small apparatus consisting of a brass ring enclosing quartz lenses. This is water cooled. The apparatus is pressed against the skin to be treated; it cools the surface and expels the blood from the skin.

Many mercury arc lamps have been invented and modified since 1835, when Wheatstone first observed that a brilliant bluish light is emitted when mercury is vaporized in an electric arc. The mercury arc produces a light rich in the shorter ultraviolet rays. Kromayer devised

a water-cooled mercury vapor quartz lamp which is used for local applications. The Burdick water-cooled lamp is similar to this. The air-cooled lamps are employed for general radiation.

One interesting modification of the mercury-vapor quartz lamp treatment is that used by Jesionek in his sanatorium at Giessen. The lamp is provided with a special reflector made of an alloy of magnesium and aluminum. The rooms are so constructed that the walls, ceiling and floor reflect the rays. The patients walk about or stand during the treatment. Several lamps are used in a room, so that a patient is irradiated simultaneously on all sides. About six patients take the treatment at a time.

The carbon arc light is the best substitute for sunlight. The open carbon arc is used considerably in lamps abroad, where it is marketed as the aureol lamp. It is much cheaper than the quartz mercury-vapor type. The clinicians at Perrysburg have also used it with considerable success.

Lamps with tungsten, iron, zinc, nickel, magnesium and other metals to form the arcs have been used. Lamps which furnish only red rays have also been marketed, but research with these has not proved satisfactory.

Methods have been provided for concentrating the solar spectrum for light therapeusis. Probably one of the earliest practical devices was described by Finsen. He used large glass lenses provided with water-cooling devices. Because of the necessary size of these, glass was substituted for quartz. The lenses absorbed the shorter ultraviolet rays but allowed those of from 300 to 380 millimicrons wave length to pass. Artault de Vevey used a biconvex lens mounted on a standard for concentrating the sun's rays. He called this a heliophore. The Thezac-Posmeur method, introduced into this country by Mrs. E. C. Post and successfully used by Lovett and Hyde and Lo Grasso, consists of a double convex lens with diameter of about 12 inches mounted in a canvas cylinder about 3 feet long. The lens is placed in one end of the cylinder which enables it to be pointed directly at the patient.

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PHYSIOLOGIC ACTION OF LIGHT

For a review of the literature on the physiologic action of light, the excellent papers of Mayer (1921) and Clark (1922) should be consulted. Some of the interesting research which has been reported since will be considered briefly here.

BLOOD

Clark (1923) measured by dialysis the diffusible calcium of blood serum in colloidin sacs against physiologic sodium chloride. The diffusible calcium was greater after the serum was irradiated by a mercury vapor quartz lamp. In rabbit serum it rose from 38 to 58 per cent, in dog serum from 44 to 53 per cent. Worringer (1923) found that the blood calcium increases when the children are irradiated by a mercury vapor quartz lamp. At the same time the symptoms of hyperexcitability disappear. The results were usually obtained within two to four weeks of treatment. The seasonal occurrence of spasmophilia is considered to be due to improper exposure to sunlight during January to May, the time when this disease is most manifest. Rothman and Callenberg (1923) have also studied the effect of quartz light therapy on the blood calcium. They noted an increase in serum calcium. In one case the calcium figure remained high for seven weeks after the last exposure. Cramer, Drew and Motham (1923) kept rats in the dark from birth. There was no apparent ill health but an abnormally low number of platelets and a slight anemia. The blood platelets increased when the rats were irradiated with a quartz mercury vapor

lamp. Pennetti (1923) found an increase in the phagocytic power of the polymorphonuclears of the guinea-pig after several hours' exposure to diffuse light.

Howe and Medlar (1924), studying the calcium and phosphorus content of the blood of tuberculous patients who were receiving Alpine mercury vapor quartz lamp therapy, found no change in the calcium and phosphorus of the blood if the concentration was normal before treatment was begun. Koopman (1924) has made a study of the effects of ultraviolet irradiation on the blood. He analyzed the blood of eight persons, five of whom were normal. The concentration of erythrocytes is not changed. The leukocyte count increases the first hour, then decreases and is still low after six hours of illumination. The neutrophil count may be increased during the first three hours. The lymphocyte count is increased. Hemoglobin, viscosity and the coagulation point are unchanged. In an experiment on a guinea-pig, Koopman found that the complement concentration was doubled during the first hour but was markedly increased during the subsequent two hours of irradiation. A three hour irradiation led to the death of the pig; the blood was free from complement. The illumination of cooled guinea-pig serum, in vitro, leads to inactivation of the complement. Mayer and Guttmacher (1924) offer some introductory work to determine what physical, chemical or physicochemical changes may take place in irradiated circulating blood (in vitro) from a bacteriologic point of view. Whatever changes are produced, they cannot destroy *Staphylococcus aureus* micro-organisms when these are mixed with blood flowing in vitro and directly exposed to the wave lengths present in the quartz mercury-vapor light radiations. If the same micro-organism is suspended in flowing saline solution and irradiated, these wave lengths are definitely bactericidal. McCaskey (1924) presents the comparative blood observations on a male patient (aged 38) after six consecutive years of quartz light therapy. He also shows the comparative result after two subsequent years, from the age of 44 to 46, when no ultraviolet therapy was used. After six years of treatment the complete blood analysis showed an increase in hemoglobin, erythrocytes, leukocytes, polymorphonuclears and a fall in blood sugar, nonprotein nitrogen, uric acid and creatinine. The carbon dioxide combining power of the blood increased from 47 to 66.2 per cent. After the two subsequent years without light therapy, the blood analysis was more like the one when treatment was commenced, except that the carbon dioxide combining power of the blood plasma had not changed. McCaskey says: "The changes produced by irradiation are eminently constructive, and belong to the plus side of the patient's vitality ledger." Laurens and Sooy (1924) studied the blood cell count of growing albino rats for the first six months of life. They found that the blood platelets decreased from a high count in rats

exposed daily to sunlight to a slightly lower one in rats exposed to diffuse daylight, and to a still lower count in rats kept in darkness. Red cells also decrease. The white count showed no constant change, but the differential counts showed an increase in polymorphonuclears at the expense of the lymphocytes. Paginez, Ravina and Sollman (1924) found that irradiation of almost any part of the body will cause a decrease in coagulation time of the blood.

Grant and Gates (1925) have shown that irradiated rabbits show an increase in blood serum calcium and phosphorus over normal values. They also found that rabbits show a seasonal tide in blood calcium, with higher calcium levels in March and December and lower blood phosphate levels. Moritz (1925) obtained results discordant with the work of Clark (1923) previously mentioned. He exposed normal rabbit's blood serum in vitro to irradiation and also irradiated the animals for two and three hours or for one hour for six days, but found no effect on the total calcium or on the diffusible calcium in vivo or in vitro. Davis (1925) explains that the blood chemical changes resulting from ultraviolet irradiation take place in two ways: first, by absorption of radioactivity by the red blood cells while they are circulating near the surface of the body on which the rays are falling, and in turn carrying the radioactivity to certain glands, thus influencing their hormone function; second, through the reflex action of the rays on certain cutaneous nerve endings which play a part in the physiologic activities of the body. Davis finds that the blood itself is not directly affected, judging from the ultimate results; however, the calcium content of the blood increases by irradiation. If it rises above normal, ultraviolet ray therapy is contraindicated. Progressive pigmentation of the skin under ultraviolet ray therapy is an index of defense against excessive changes in the chemistry of the blood.

Miles and Laurens (1926) studied the effect of light and darkness on some of the physical characters of the blood of dogs. Subjecting the animals to a short period of darkness results in an initial decrease in red blood cells, which returned to normal during the dark period. When the dogs are readmitted to light, the values fluctuate above and below normal before a level is maintained. Under similar circumstances the white blood cells at first increase, but there are slight fluctuations before the normal is attained. The differential count shows slight changes; platelets behave like the red cells. The return to light causes a temporary increase; coagulation time is also increased. Carbon arc irradiation with moderate dosage (1 hour per day for eight days) results in an initial increase in red blood cells followed by a decrease; with increase of dosage (2 hours per day for eight days), an immediate decrease, and with both dosages, a gradual increase to a new level. They believe this indicates a stimulation of the hematopoietic organs which is at first

obscured by increased blood volume. The total solids and hemoglobin decrease, but return to normal shortly after the irradiation period is ended. White blood cells show fluctuations above the normal level, but soon return to normal following irradiation. Irradiation increases the polymorphonuclears at the expense of the lymphocytes; platelets increase with moderate dosage, decrease with stronger dosage and attain values above normal in the postirradiation period. Reed and Tweedy (1926) found that irradiation of blood in vivo with a carbon arc lamp does not produce any noticeable change in the blood calcium of dogs under ether anesthesia.

Blood Pressure.—Reed (1923) reported that the exposure of the eyes of dogs, anesthetized with chloretone, to light of approximately 100 faradic current intensity from a 1,000 watt nitrogen tungsten projection lantern, resulted in a transient depression of blood pressure. Reed (1925), carrying his experimentation further, concluded that a beam of light of approximately 100 faradic current intensity directed into the eyes of a dog under ether anesthesia will cause a pronounced depression of arterial blood pressure. A more intense light (1,250 faradic current) produces a more profound effect, but the change is not of proportionate magnitude. The effect is not due wholly to nervous factors, for bilateral vagisection and section of the optic nerve do not abolish or prevent the effect, although the latter operation modifies it considerably. Since no evidence was obtained that photochemical decomposition had liberated any depressor substance into the blood, it seems that the mechanism must be due to the effect on the blood. Direct irradiation of blood circulating through a quartz tube placed in an artery will cause a great depression of general arterial blood pressure. Animals sometimes die from the shock even when anesthesia has been discontinued. The effect is obtained by irradiation with a carbon arc rich in ultraviolet or with the infra-red ultraviolet filtered out of the spectrum. Since irradiation decreases the coagulation time, to keep the quartz tube clean it was necessary to use heparin which modified the result. Aimes (1925) has observed an elevated blood pressure by irradiation.

METABOLISM

Cori and Pucher (1923) found that roentgen-ray therapy produced a marked increase in urinary nitrogen and phosphates. Barkus and Balderrey (1923) showed that sunlight or the light of a mercury-vapor quartz lamp caused disodium hydrogen phosphate, injected into the blood of sheep, to leave the blood more rapidly. This they claimed was due to increased glomerular filtration or to the dilatory action in the peripheral vessels.

Degkwitz (1924) prevented the development of rickets in puppies kept in the dark by giving them vigorous daily exercise. The puppies

in the dark weighed more than the control puppies kept in the light. Histologic examinations revealed no pathologic changes. A chemical analysis of the organs and tissues showed a distinct impoverishment of calcium, phosphorus, sodium and magnesium, and an increase of potassium. The nitrogen retention was lower in the animals kept in the dark. This was especially true of the animals that were fed a rich protein diet. Since the experimental animals were in as good condition as the control animals kept in the light, Degkwitz thinks that the mechanism is that of adaptation. Wiener (1924) irradiated men with a carbon arc lamp which has almost a continuous spectrum from 400 to 290 millimicrons. The men showed a negative nitrogen balance which became positive with exposures of thirty to 100 minutes. There was a phosphorus retention; sulphates showed no variation. Turpin, Zizine and Lesné (1924) exposed three rats, aged 15 days, fifteen minutes daily for thirty days to the light of a mercury vapor quartz lamp. Analyses showed that the total calcium content of the irradiated animals was appreciably greater than that of the control animals. Hart, Steenbock and Elvehjem (1924) studied the effect of irradiation on the calcium and phosphorus equilibrium in mature lactating goats on negative calcium and phosphorus balances. One animal was irradiated by a mercury vapor quartz lamp (distance, 2 feet) ten minutes daily for a week, then the exposure was increased to twenty minutes for six weeks more. The negative calcium balance gradually changed to a positive balance although the intake remained the same. Analyses of the blood serum at the end of the experiment showed a marked increase in phosphorus but no change in calcium. Similar results were obtained in another goat irradiated twenty minutes daily for four weeks. A third nonlactating goat which was irradiated thirty minutes daily for three weeks showed an increased phosphorus retention in the blood. In one animal there was a slight increase in the urinary phosphorus and a decrease in the fecal phosphorus; in the other animals the excretion of phosphorus was only slightly affected. Orr, Magee and Henderson (1924) fed two pigs, which were kept in semidarkness, on a diet high in phosphorus and low in calcium. One pig, placed 3 feet from a carbon arc lamp, was irradiated daily for twenty-four days. It showed an increased calcium and phosphorus absorption and retention. Orr, Holt, Wilkins and Boone (1924) irradiated children and obtained similar results. Before irradiation, less than one half of the phosphorus was excreted in the urine; after irradation, the amount increased to from 50 to 75 per cent. This was the result of decreased elimination in the stool, not to an actual increase in the urinary elimination. The calcium partition was not constantly affected. When Orr and his co-workers injected calcium subcutaneously into a rachitic child, most of the calcium was excreted by the intestine; if phosphorus salts are injected, most

of them are recovered in the urine. They conclude that since the calcium in the intestinal contents is largely bound with phosphorus, apparently these two elements are absorbed together, and that the irradiation increases absorption rather than decreases the reexcretion. Crofts and Laurens (1924) investigated the influence of light on the respiration of the frog. They found that the absence of light disturbs the equilibrium just the same as a short exposure to irradiation. If the frog is kept in the dark for a long period, the equilibrium is adjusted, but on readmission of light it is again disturbed.

Henderson (1925) carried on experiments similar to those which Orr and his co-workers (1924) made on pigs, but he used a better balanced ration ($P_2O_5 : CaO = 1 : 1$). He found that it was not possible to stimulate an already positive balance. Unfortunately, the control animals developed a pathologic condition during the experimental period. Maynard, Goldberg and Miller (1925) pointed out that the ash content of pigs exposed to the sunlight in the spring is greater and that there is an absence of stiffness of the legs, compared with a series of animals exposed during the winter. Mayerson, Gunther and Laurens (1926) subjected normal adult dogs on an adequate diet to short periods of darkness (from eighteen to twenty-eight days) which they found to act as an initial stimulus to metabolism, decreasing to normal at the end of the period. There is an increase in total acidity, an increased nitrogen and chloride excretion in the urine, a rise in blood phosphorus and a retention of calcium and phosphorus. Admission of light results in a second similar increase in metabolism which lasts a short time. Irradiation of the dogs increases the endogenous nitrogen metabolism, stimulates the absorption of calcium and phosphorus from the intestine, and decreases the blood sugar. These changes persist and are sometimes more apparent after irradiation has ceased. Serum phosphorus is markedly increased by irradiation, in some cases 300 per cent. Moderate exposure to the arc lamp brings about a balance between the serum calcium and phosphorus. Excessive or repeated irradiation will increase the blood serum calcium.

PIGMENTATION

Bang (1924) has advanced a theory regarding the pigmentation of the skin during heliotherapy. He investigated the division of the bactericidal rays of the spectrum of a mercury vapor quartz lamp, and found them sharply limited to the same wave lengths as Hauser and Vahle found for pigmentation; namely, 300 millimicrons. Bang considers that if this is true, pigmentation must be connected with an important cellular process occurring in the deep-lying epithelial cells of the epidermis. The pigment has its source in the blood which furnishes the pro-pigment, and in a ferment found in the epithelial cells

where the pro-pigment becomes melanin (the pigment) by oxidation. The pigmenting action of light seems closely connected with other vital processes in the cells, since a given wave length has both a definite pigmenting, bactericidal and blood destructive action. Jansen (1924) does not agree with one of Bang's assumptions; namely, that heliotherapy is most effective in people who pigment abundantly. Jansen believes the significance of the pigment is its protective qualities against light and especially the ultraviolet rays. As a proof that pigmentation is not the deciding factor as to the benefits derived from heliotherapy in tuberculosis, the author cites the fact of the intense pigment produced by the mercury quartz lamp treatment as contrasted with the golden-brown skin development in mountainous regions. The beneficial results are much greater with direct exposure to sunlight at higher altitudes, and are not dependent on the amount of pigment in the skin. In view of these observations, the frequency of rickets in negroes may be due to an inability of the ultraviolet rays to penetrate the pigment layer, so that their beneficial influence on the mineral metabolism is lost.

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HELIOTHERAPY AND ACTINOTHERAPY IN VARIOUS DISEASES

The use of light in the treatment of disease has been applied in many branches of medicine. Those of most interest to the pediatrician, namely, tuberculosis and rickets, will be considered in detail. Some of the others which do not have such an extensive literature will be mentioned only.

Clark (1925) showed that ultraviolet irradiation is ineffectual in preventing scurvy in guinea-pigs fed with different scorbutic diets. Armand-Delille and Linossier (1925) report three cases of athrepsia, in which the patients showed marvelous cures with gain in weight and normal development when the infants were irradiated. Lucas (1920) recommended heliotherapy for the treatment of anemia, believing that light acts as a stimulus to the blood forming organs.

Phototherapy has proved useful in dermatology. Sequeira (1924) summarizes some of the diseases the dermatologist treats by irradiation; namely, psoriasis, keratoses, leprosy, granulomas, scars, ringworm and carcinoma of the skin. Hawks (1917) used ultraviolet light extensively for diseases of the skin, open wounds, general debility, gout, lumbago and rheumatism. Costello (1925) irradiates the thymus in cases of infantile eczema. Porcelli (1925) and Mosse (1924) also irradiate patients with eczema. Bryant (1918) suggests ultraviolet light for curing boils.

TUBERCULOSIS

The first systematic study of heliotherapy in the treatment of tuberculosis was made by Finsen, who was the inventor of the special application of light rays in the treatment of lupus. In 1895, Finsen founded the Light Institute in Copenhagen for the treatment of lupus and other diseases by light. Benard, in 1902, in the sunny Engadine valley of Switzerland, found that sluggish wounds which were difficult to cure healed rapidly when they were exposed to the intense sunlight of that

altitude for several hours daily. Encouraged by the results obtained with wounds, he was equally successful in treating local tuberculosis and specific ulcers with sunlight.

In 1903, Rollier founded his famous clinic at Leysin for the treatment of surgical tuberculosis in adults and children with heliotherapy. So great has been his success that there are now three sanatoriums instead of one. To Rollier more than to any one else is owed the development of heliotherapy as a clinical aid. He has shown that a large number of surgical cases can be cured without operation. Rollier has dealt mostly with extrapulmonary tuberculosis. His opinion regarding pulmonary tuberculosis is as follows:

> To treat it adequately is beyond our power as our experience has been almost confined to surgical manifestations of the disease; properly applied, I am convinced that heliotherapy would be a useful factor in the treatment of the great majority of cases of pulmonary tuberculosis. Heliotherapy has not yet won its spurs in the treatment of pulmonary tuberculosis; its results have, none the less, been sufficiently good to encourage our colleagues, whose experiences in this disease are greater than our own, to make a serious attempt to apply systematized sun treatment to suitable cases under their care.

Heliotherapy can be carried out in any place where there is sunlight, and the benefits derived from its use are well illustrated by the old Italian proverb which states, "Dove non va il sole, va il medico" (Where the sun does not enter, the doctor goes). Both Rollier and Finsen were convinced that heliotherapy at a high altitude was most beneficial. Jaubert (1913) believes that the best results from altitude are obtained at an elevation of from 2,000 to 3,000 feet. Rollier says:

> To reach sea-level the sun's rays have to pass through the whole thickness of the atmosphere; over all our big cities the latter is rich in mists, dust and micro-organisms of every kind, which absorb both heat and light rays in a proportion sometimes as high as 95 per cent. This absorption not only deprives the patient of a large number of useful rays, but by heating the air may give the sun-bath a relaxing instead of a stimulating effect. At high altitudes, on the contrary, the air is transparent, free from solid particles, and easily traversed by the sun's rays, which pass through without absorption, and warm and invigorate the body of the patient while leaving the air cool and fresh. For this reason a much greater intensity of direct sunlight can be borne at high altitudes, and heliotherapy may be practiced in every month of the year.

Two interesting reports of treating tuberculosis at high altitudes have recently been published by Neuman and Amrein. Neuman (1924) has written on the characteristics of the high Alpine climate and cure at Davos. Davos is situated in an elevated valley at an altitude of from 5,000 to 6,000 feet above sea-level. It was the first place in the high Alps to be developed as a health resort for tuberculous patients. Neuman states that Turban's statistics covering a period of ten years showed that the results obtained by the combined effects of Alpine climate and careful

sanatorium treatment far surpassed any obtained elsewhere. One half of his patients were, according to the Turban-Gerhardt classification, in the second stage, and a fourth in each of the first and third stages. Turban claimed that 66.1 per cent of all these cases were restored in the sense of Brehmer's absolute or relative cure. Of these cases of arrest, 99.1 per cent were cases of the first, 73.2 per cent of the second and 23.6 per cent of the third stage of tuberculosis of the lungs. Amrein (1924) writes about the climate of Arosa, situated at a height of 7,000 feet, for tuberculous patients. Arosa offers all the chief properties of the climate of a high altitude; namely, low barometric pressure, dryness and purity of air, low temperature and a great deal of sunshine. The best known physiologic effects caused by this high altitude are increase of the red blood corpuscles, the lymphocytes and the large mononuclear cells, decrease of the granular leukocytes, and increased viscosity of the blood. Respiration is slower and deeper; circulation is improved. The following types of pulmonary tuberculosis are contraindicated for Arosa: advanced cases with a permanent pulse rate of 130 and more; persistent temperature of 101.5 F.; higher extensive involvement of the larynx, kidneys and intestines; emphysema; noncompensated valvular lesions of the heart; marked arteriosclerosis; diabetes; gout; anemia and psychoneurosis. Bacmeister and Baur (1922), Hill (1923), Kelynack (1923) and Hudson (1923), all favor a high altitude with plenty of sunlight for the treatment of tuberculosis. Schmerz (1914) reports favorable results in treating surgical tuberculosis by sunlight in the lowlands.

Rollier has developed a definite technic for the use of heliotherapy which he thinks gives the best results. The individual differences in the adaptability of patients should be considered carefully. Care should be taken to avoid chilling and unnecessary exposure to winds. Exposure always begins with the feet, legs, thighs, abdomen and thorax, following in the order named, with an interval of a varying number of days between each. Only a few minutes' exposure to sunlight is allowed to each part of the body on the first day of treatment. The length of the exposure is gradually increased, until the patient is able to expose his whole body to the sun for from several hours to almost all day in either summer or winter. The head is usually covered or kept out of the sun. Increased precautions are necessary in cases of pulmonary and abdominal tuberculosis and secondary infection without free drainage. These can, however, be greatly benefited if the technic is properly carried out. Rollier regards plaster casts as a great hindrance to heliotherapy. He replaced the cumbersome plaster devices with simple arrangements of webbing straps which permit free access of air and sun, complete immobilization and adequate extension.

The technic employed by various investigators is practically the same. Rollier's probably is used most frequently. The length of exposures

varies with different clinicians. Malgat irradiates for twenty minutes, Bernhard for three or four hours, Szaboky for two hours a day and Rollier for many hours daily. "It has been suggested that heliotherapy is more effective with children than with adults; I (Rollier) have always had a considerable majority of adult patients and have not been able to support this view, except, perhaps in certain synovial infections of the knee and in genito-urinary tuberculosis." Rollier's results, obtained from 1914 to 1921, for osteo-articular tuberculosis only may be seen in table 1.

TABLE 1.—*Results of Heliotherapy in Rollier's Patients from 1914 to 1921*

	Children		Adults		Total	
	Number	Per Cent	Number	Per Cent	Number	Per Cent
Cured	681	86.5	1,065	77.2	1,746	80.5
Improved	51	6.5	191	13.8	242	11.2
Stationary	43	5.5	104	7.5	147	6.8
Worse and deceased	12	1.5	20	1.5	32	1.5
	787		1,380		2,167	

TABLE 2.—*Results of Heliotherapy in Rollier's Patients Preceding 1914*

	Number	Per Cent
Cured	945	83.7
Improved	112	9.9
Stationary	41	3.6
Deceased	31	2.8
Patients	1,129 (662 adults, 477 children)	

Statistics up to 1914, including all types of surgical tuberculosis, are given in table 2.

Rollier has obtained permanent cures in those patients who work in suitable surroundings after leaving the sanatorium. For children, he recommends an open-air school in which the sun cure can be continued; for adults, agricultural work is desirable. Surgical and pulmonary tuberculosis are, in many cases, the result of a secondary infection of the tracheobronchial or mesenteric glands. The infection usually occurs in childhood and may be latent for a long period. For delicate or predisposed children, Rollier recommends as much fresh air as possible day and night, plenty of good simple food and school work reduced to a minimum. In summer, the children wear only bathing suits, and in winter as few clothes as possible. A great deal of attention is given to Swedish drill, which teaches proper chest expansion and nose breathing. Prophylactic measures along these lines eradicate the disease completely in almost all cases.

Revillet (1912) obtained excellent results with heliotherapy in treating children suffering from surgical tuberculosis. Some Swiss children were treated at Revillet's sanatorium. They were examined before and after treatment by d'Espine, thus checking the results. The diseased area was exposed to the sun for one-half hour to an hour at first, and finally for two to three hours or all day. The children were given sea baths lasting from one to three minutes daily. The results were striking: Of sixty-three children suffering from Pott's disease, twenty-seven were cured, twenty-six were improved, six remained stationary and four died; of ninety-four suffering from hip disease, thirty-three were cured and fifty were improved; in seven cases of tuberculous peritonitis, three were cured, three were improved and one died; in eleven cases of lupus, eight were cured and three were improved.

D'Oelsnitz (1912) reports three cases of tuberculous peritonitis with ascites in which the patients were cured in one, three and five months, respectively, by heliotherapy. Poncet and Leriche (1912) recommend laparotomy and heliotherapy for ascitic tuberculous peritonitis. They consider that better and more lasting results are thus obtained than by the use of either measure alone. Pottenger (1915) and Kime (1916) supplement the exposures to sunlight by directing concentrated light on the chest by means of a blue concave mirror.

Campbell (1916), who has successfully used heliotherapy in the treatment of bone and joint conditions, states that the most interesting features about heliotherapy are: 1. Sequestrums are rapidly expelled. 2. Marked and early beneficial effects are obtained in severe septic conditions. 3. There is a rapid evolvement of tuberculous processes resulting in bone ankylosis, not the mobilizing action Rollier speaks of. 4. Orthopedic corrective measures are as necessary as before the use of light.

Campbell has found that the length of time required to procure results is much shorter than Rollier thinks necessary to effect a cure. Bordier (1916) considered that he obtained the best heliotherapeutic results by using a cloth screen of specific thickness in front of the patients to be irradiated and in the windows of his solaria. Kisch (1919) combined heliotherapy with passive hyperemia and the administration of sodium iodide for treating surgical tuberculosis. His reports show excellent results in cases of severe tuberculosis of the joints. Schwyzer (1920) thinks the chief objection to heliotherapy is the length of time required to cure a patient; therefore it is better to use a combination of surgery and heliotherapy. Schimer (1920) agrees with Schwyzer, and considers that artificial heliotherapy is most desirable.

Hyde and Lo Grasso (1921) have successfully used Rollier's technic at Perrysburg, N. Y. Their results are similar to Rollier's, but are not so favorable. It is possible that the lower altitude might account for the

difference. On dark days they use the mercury-vapor quartz lamp, but consider it inferior to sunlight. This is the consensus of opinion of most clinicians. The mercury vapor lamp treatment has been useful in treating intestinal tuberculosis. In pulmonary tuberculosis, natural and artificial heliotherapy are likely to cause hemorrhages. It is possibly the result of congested lungs which are known to occur after animals are exposed to ultraviolet light (Levy, 1919, and Gassul, 1920).

Hill, Campbell and Gauvain (1922) have studied the metabolism of children crippled with surgical tuberculosis. They found an increase of 40 per cent above standard for children of the same weight confined in a closed calorimeter. After three months of treatment of graduated exposure to open air and sunlight, the child's metabolism taken out of doors was 20 per cent above that taken in a well ventilated cubicle soon after admission to the institute. Children who were treated for many months showed a high metabolism in the outdoors condition. Those who were well pigmented gave about the same figures as those who did not pigment well. It is considered that the rise in metabolism caused by heliotherapy per se is insignificant compared with that caused by exposure to open air. Gassul (1921) has published a monograph which gives a summary of the treatment of tuberculosis by heliotherapy (natural and artificial), roentgen ray and radium. He obtained a high percentage of cures in all kinds of surgical tuberculosis by the use of irradiation.

Graf (1922) combined heliotherapy with tuberculin in treating sixty-one cases of surgical tuberculosis. Most of the results were negative; however, good results were obtained in a few difficult cases. He discusses the indications and contraindications for the use of tuberculin and sun treatment.

Lucas (1920) using heliotherapy for peritonitis, obtained better results when an operation was performed first and then sunlight used as soon as the distention was relieved.

Myll (1924) thinks that heliotherapy arrests the progress of pathologic change, and seems to promise a cure of tuberculosis of the genito-urinary tract. Girdlestone (1924) says that operative procedures are seldom, if ever, warranted in children under the age of 16 suffering from surgical cases of tuberculosis. Rest, good food, open air and sunshine are essential therapeutic measures. Butler (1924) could not improve cases of lupus erythematosus by ultraviolet rays, but obtained favorable results with carbon dioxide snow or radium therapy.

Lo Grasso (1924) has written extensively on the scientific treatment of tuberculosis. The article deals with the treatment of extrapulmonary tuberculosis as carried out at Perrysburg. It is essentially the technic of Rollier, with such variations as have been considered advisable owing

to peculiar local conditions. In the summer the middle of the day is contraindicated, but that period is most desirable in the spring and fall. On admission, the patient is gradually accustomed to an outdoor life before exposure to the sun is begun. Patients with fever or nephritis must be watched carefully. Sun baths are discontinued an hour before and for two hours after the midday meal. Chilling may cause irreparable harm, therefore care must be taken that no breeze strikes the body. After each sun bath the patient is given a sponge bath; if the skin is very sensitive, a vegetable oil such as olive or coconut oil is applied. The following is a modification of Rollier's technic as employed by Lo Grasso: First day: The patient, reclining in bed with the eyes protected from the sun by sun glasses and the head by a cap, is taken to the sun-porch. Trunks only are worn, and blankets or sheets are used for covering, depending on the season. Only the feet are exposed to the sun for five minute periods, three times during the day, with an hour interval between the periods. Second day: The period of exposure for the feet is increased to ten minutes, and the knees to the ankles are treated for five minutes. Third day: The time of exposure for previously insolated parts is increased by five minutes, and the lower extremities from the knees to the hips are exposed to the sun's rays for five minutes. On the fourth and fifth days the exposure for each part is increased by five minutes, and the abdomen on the fourth and chest on the fifth day are exposed for five minutes and daily increased by the same period. On the twelfth day the part which has been exposed the longest receives a total irradiation of three hours; the periods of exposure for the feet is sixty minutes; knees to ankles, fifty-five minutes; thighs, fifty minutes; abdomen, forty-five minutes and chest forty minutes. From this until the sixteenth day, the period of exposure for the feet remains sixty minutes. The remaining divisions are increased by five minutes each day until each subdivision receives sixty minutes exposure at a time. On the seventeenth day the insolations are reduced to one morning and one afternoon period of ninety minutes each. If necessary, the period of exposure may be shortened or gradually increased to four hours daily. The total daily exposure should be divided equally between the anterior and posterior parts of the body. If the patient cannot be turned, the time is awaited until the physical condition will permit turning. During insolation, ulcers are covered only by a wire screen; in the evening they are cleansed with alcohol and a dry dressing is applied. If it is necessary for any reason to interrupt the insolation, it is resumed at an earlier stage than that at which it was stopped. Sometimes a simpler method is used in which the whole body is exposed from the first day. Head and eye protectors are used as in the previous method. During the first day the body is exposed for three morning and afternoon periods of two minutes at hour intervals. The exposure

period is increased daily for fifteen days, two minutes each day until the total period of irradiation will have reached three hours. The number of exposures is reduced to two in the morning and two in the afternoon, with hourly intervals. On days when the sun bath is not feasible, an air bath is given. The progress of patients is in direct proportion to the intensity of pigmentation. Of the 720 patients with extrapulmonary tuberculosis discharged between 1913 and 1923, 78 per cent of the adults and 18 per cent of the children showed a clinical pulmonary lesion. The results in these cases seem to warrant the use of heliotherapy in pulmonary tuberculosis. Surgical interference is not so satisfactory as heliotherapy, and is justified only in those cases in which the local lesion has not shown any satisfactory improvement. Lo Grasso considers artificial light therapy a poor substitute at best for sunlight.

Knowler (1924) has substituted heliotherapy for the thermocautery, the classic treatment of tuberculosis of the epiglottis, with some success.

Gerstenberger and Wahl (1924) made a study of ultraviolet ray therapy in peritoneal and glandular tuberculosis of children. Their observations were made in ten cases of various forms of tuberculosis treated by the Alpine lamp. They conclude: The sole use of ultraviolet rays, as produced by the quartz mercury-vapor lamp, has been of decided value in the treatment of peritoneal, lymphatic and osseous tuberculosis. Of the lymphatic forms of tuberculosis, the mesenteric is most rapidly improved, next the mediastinal, and last the peripheral. Pulmonary tuberculosis of the miliary type remains wholly uninfluenced by ultraviolet ray therapy, even though it is begun in the course of the disease. Pfefferkorn (1924) has also used artificial light for treating peritoneal and mesenteric lymph node tuberculosis with good results. In children, the best results were obtained in the exudative forms, but it was efficacious in the dry adhesive forms of children as well as in the peritoneal tuberculosis of adults. Seven per cent became worse, with fever and ascites, 7 per cent died, 9.3 per cent were slightly improved and 76.8 per cent were definitely improved.

Pattison (1924), discussing the therapeutic effects of ultraviolet irradiations, states that all the benefits derived from insolation are not due to the effect of light, but that part are the result of exposing the body to cool air which has been shown to raise the basal metabolism markedly. He attributes the beneficial effects to an increase of the bactericidal power of the blood and an activation of the vitamins in the body.

Saidman (1924) reports on 200 cases of tuberculosis and about 3,500 exposures. He considers two principal reactions of an irradiated body, actinic erythema and pigmentation. He says after an actinic

erythema the section of a fistula may be checked, pains relieved and the general state improved. Erythema is avoided in cases of pulmonary tuberculosis, serous peritonitis and in the presence of fever. The best results are obtained in cutaneous localization, cold abscesses, adenitis, osteo-arthritis and fistulous lesions in general. In cases in which improvement is noted for a few weeks, only a combination with roentgen-ray therapy may yield good results. The roentgen rays exert a local action, the ultraviolet a general action, and the infra-red an analgesic and hyperemic action. Recent forms of adenitis do not always respond. If caseating forms are treated by radiotherapy alone, they often ulcerate, but the combined treatments avoid this. Dry forms of peritonitis yield good results. For cutaneous lesions, the high frequency electric current may also be used. By the use of these other measures, Saidman avoids the absolute dependence on weather or climate for treatment.

Robertson (1924) is enthusiastic about quartz lamp therapy in cases of tuberculous cervical adenitis, lupus and tuberculids. He uses the technic of Baeh, which it might be well to mention at this time. The lamp used was run on 250 volts direct current with a resistance, so that 140 burner volts were produced with 2.5 current amperes. Daily exposures starting with three minutes at 39 inches distance were first used. These were increased three minutes each day, and the distance was diminished by 4 inches at short intervals until thirty to forty-five minutes at 20 inches was reached. The eyes were protected. The penetrating powers of the rays were increased by painting the skin with a colloidal silica ointment. The mechanism which produces the beneficial results is unknown. Robertson suggests that it may be due to the effect on the general metabolism through the action on the skin which produces phagocytosis and increased blood change.

Hill (1924) discusses the value of heliotherapy for pulmonary tuberculosis. He says that the sun treatment has fallen into disrepute and is considered dangerous by experienced medical men. This is largely due to the manner in which the treatment is given. The treatment has been given in sun boxes, and febrile patients have been subjected to conditions which overheat instead of cool the body. This form should not be used indiscriminately for pulmonary tuberculosis. It should be given in the open air and the excessively hot sun avoided. Heliotherapy can be used for tuberculosis of the lungs when the disease is of the simple focus type which approximates surgical tuberculosis in character. Good results have been obtained in patients with the pleural type, the bronchial asthmatic type and in those who have no fever and the activity of the disease has been arrested. Sun treatment for pulmonary tuberculosis is contraindicated in cases with smoldering activity of the tuberculous process, with a tendency to autoinoculate on exertion, to hemoptypsis,

and to other signs and symptoms of activity. Fecht (1924), studying 356 cases of pulmonary tuberculosis during quartz lamp irradiation, has worked out a basis of prognosis. He determines the normal leukocyte and differential counts of the patient on admission. After three weeks treatment, the blood is again analyzed. The prognosis is judged on the basis of the lymphocytosis, normal lymphoid cell count or lymphocytic decrease in relation to the Arneth scale, as good, fair or poor. Mayer (1924) does not encourage the use of light for the treatment of pulmonary tuberculosis. He advocates it as an adjuvant in bone, joint, peritoneal, fistulous and lymph node tuberculosis. He suggests that the future possibilities of light therapy will be greater cooperation between the clinician, the physicist, and the laboratory experimentalist.

Erickson (1925) reports that in a series of eighty-one cases of intestinal tuberculosis complicating pulmonary tuberculosis, there was a favorable result in 85.2 per cent following the use of ultraviolet light as judged by complete relief or definite improvement in symptoms, and there was no result or an unfavorable one in 14.8 per cent. Of these cases, in 24.7 per cent there was entire relief of symptoms; great improvement in 47 per cent, and definite though less improvement in 13.5 per cent.

Hall (1925) considers that ultraviolet light is a potent remedy for pulmonary tuberculosis and that both adults and children do well when thus treated. Too rapid pigmentation is to be avoided, as it is protective against further dosage and prevents the "provocative reactions" which, if carefully judged, are followed by an increased immunizing response. McCutcheon (1925) considers heliotherapy most valuable in the treatment of pulmonary tuberculosis. He finds the most remarkable effect on pulmonary cases to be the decrease in lung moisture. Gibson (1925) advocates sanatorium treatment for juvenile patients with tuberculosis when heliotherapy can be employed.

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(To be continued)

News and Comment

MEETING OF CENTRAL STATES PEDIATRIC SOCIETY

At a meeting of the Central States Pediatric Society, held in St. Louis, October 22-23, the following officers were elected: Dr. O. L. Goehle, Cleveland, president; Dr. Fred H. Moore, Des Moines, Iowa, vice-president; Dr. H. T. Price, 121 University Place, Pittsburgh, secretary-treasurer; executive committee: Dr. O. N. Torian, Indianapolis, Dr. C. G. Grulee, Chicago, and Dr. Elmer T. Timmons, Colorado Springs, Colo.

MASSACHUSETTS

Joint Meeting of Pediatricians.—The New England Pediatric Society, the Philadelphia Pediatric Society, and the Section of Pediatrics of the New York Academy of Medicine held a joint meeting in Boston, October 16. There were papers by Dr. Bengt Hamilton on "Observations on the Acid Metabolism of Intoxicated Infants," and by Dr. George M. Guest on "The Variations in the Diastase of the Blood of Infants." There was a lecture by Dr. Elliott P. Joslin at the Deaconess Hospital on "Diabetes in Childhood." Luncheon was held at the Harvard Medical School, and Dr. Harvey Cushing discussed "Neurological Surgery in Childhood" at the Peter Bent Brigham Hospital in the afternoon.

NEW YORK

Personal.—Dr. Adolph G. G. De Sanctis has been appointed professor of pediatrics at the New York Post-Graduate Medical School and Hospital.

PHILADELPHIA

Anniversary of Pediatric Society.—At the thirteenth anniversary of the Philadelphia Pediatric Society, November 9, Dr. J. P. Crozer Griffith delivered the Frederick A. Packard Memorial Lecture on "Some Neurotic Disorders in Children." Dr. Griffith, the founder and first president of the pediatric society, was guest of honor at a dinner at the Rittenhouse Hotel preceding the meeting.

VIRGINIA

Pediatric Society Organized.—The Virginia Pediatric Society was organized during the recent annual meeting of the Virginia Medical Society at Norfolk, for the purpose of advancing the cause of pediatrics in Virginia. Dr. Nathaniel Thomas Ennett, Richmond, was elected president; Dr. Lawrence T. Royster, University, vice-president, and Dr. William B. McIlwaine III, Petersburg, secretary-treasurer.

Obituaries

E. CHARLES FLEISCHNER, M.D.

1882-1926

Dr. E. Charles Fleischner was born in New Haven, Conn., in April, 1882, and died, Oct. 11, 1926.

He graduated from the medical school of Yale University in 1904, and served internships in the New Haven Hospital, the Seaside Hospital, St. John's Guild, New York, and also at the Nursery and Child's Hospital, New York. During 1906 and 1907, he studied in foreign clinics. He established himself in San Francisco in 1909, and acted as instructor in pediatrics at the Leland Stanford University until 1913. At this time he accepted an instructorship in the department of pediatrics of the University of California Medical School, and at the time of his death he held the chair of clinical professor in this school. He was a member of the national societies which are devoted to pediatrics, bacteriology and the more purely scientific branches, as well as being an active participant in the state and local societies. During 1918, 1919 and 1920, he was secretary of the section of Diseases of Children of the American Medical Association, and in 1921 he was Chairman.

He contributed a series of bacteriologic studies on *Bacillus abortus* and *Bacillus melitensis,* dealing with the pathogenicity of these organisms in monkeys.

He published several articles on the treatment and control of diphtheria. Shortly before his death he was engaged in an intensive study of the treatment of poliomyelitis with convalescent serum. In this problem he was intensely interested, and he devoted himself with great industry and enthusiasm to its solution. He also wrote a number of clinical papers, and in collaboration with Dr. William Palmer Lucas he contributed a complete monograph on the diseases of the blood to the recent System of Pediatrics.

Dr. Fleischner was endowed with a splendid intellect. He saw things clearly, and he had the ability to express himself with directness and precision. He was not only a skilful worker in the laboratory, but he was also a keen and accurate clinician. Students found in him an inspiring and stimulating teacher.

No less striking than these qualities were his high standards of manly honor and professional integrity. He always had the courage of his

convictions and the courage to do right in all his relations as physician and citizen. The warmth of his friendship was received and reciprocated by his colleagues in every part of the country.

It may be said truly that in the death of Dr. Fleischner medicine as a whole, and pediatrics in particular, have sustained a great loss.

I. A. A.

GEORGE M. TUTTLE, M.D.
1866-1926

George Marvine Tuttle, son of Daniel Sylvester Tuttle and Harriet Minerva Foote, was born in Morris, New York, in 1866.

He devoted the major part of his professional life to pediatrics, and in this specialty he became eminent as a practitioner and as a teacher. On the death of Dr. Luedeking, Dr. Tuttle became professor of pediatrics in Washington University School of Medicine, and when this school acquired control of the St. Louis Children's Hospital he was made chief of staff. Later, when the school was reorganized on a full time basis, he became consultant to the staff and clinical professor of pediatrics, positions which he filled with honor to the school and to himself until his death.

Dr. Tuttle was essentially a clinician, and wrote a book on pediatrics which reflects the soundness of his clinical mind. He was a most helpful member of the St. Louis Pediatric Society and several times served as its president; he was president of the Central States Pediatric Society at the time of his death.

The news of Dr. Tuttle's death came as a shock; the fact seems to be sifting slowly into our minds. He was in London, at the end of his vacation and was about to leave for home when, without warning, he became acutely ill and passed away before medical aid could reach him.

He is survived by Mrs. Tuttle and a son, Wallace.

A. B.

Abstracts from Current Literature

Anatomy and Physiology

EXPERIMENTAL STUDIES ON THE SUPRAVITAL STAINABLE SUBSTANCES IN THE ERYTHROCYTES. MUNEO NISHITANI, Oriental J. Dis. Inf. **1**:83, 1926.

The author has made a biologic and microscopic study of stainable substances seen after the vital staining of erythrocytes. These substances occur in several different forms, such as filamentous and coarsely and finely granular. They are usually stained only by basic staining dyes. Certain poisons, such as lead and phenylhydrazine, cause an increase in their number. They are present in both immature and normal erythrocytes. Injection into rabbits of Heinz's corpuscles — a stainable body obtained in erythrocytes by action of hydroxylamin — led to a blood picture and organic changes similar to those seen in pernicious anemia.

The author concludes that the freeing of these supravital stainable substances in the blood acts as a toxin to the blood and blood-producing organs, and tends to act unfavorably in anemia.

HIGGINS, Cincinnati.

Pathology, Bacteriology and Biochemistry

THE LIVER AS A SITE OF BILIRUBIN FORMATION. F. C. MANN, C. SHEARD, J. L. BOLLMAN and E. J. BALDES, Am. J. Physiol. **77**:219, 1926.

The problem of bilirubin formation in the liver is complicated by the fact that in its dual blood supply one element, the portal blood, flows in part directly from an organ which itself is the site of active bilirubin formation — the spleen — as well as by the fact that the liver is the most active site of elimination of this substance. These difficulties were overcome by removing the spleen, and preventing elimination by removal of the gallbladder and ligation of the common duct. Dogs treated in this way then had a uniform bilirubin content in all blood entering the liver identical with that of the general arterial circulation.

With this procedure there was found an altogether unexpectedly rapid rise of bilirubin following removal of the gallbladder and obstruction of the common duct. The bilirubin content of the portal blood was the same as that of the arterial blood, but in blood withdrawn from the vena cava after leaving the liver there was a definite, constant increase of bilirubin. That this was not due to temporary accumulation of bilirubin in the liver cells, with subsequent liberation, was indicated by its constancy at all stages of the experimental procedure.

H. E. EGGERS.

INSENSIBLE PERSPIRATION: ITS RELATION TO HUMAN PHYSIOLOGY AND PATHOLOGY. F. G. BENEDICT and H. F. ROOT, Arch. Int. Med. **38**:1 (July) 1926.

Benedict and Root's excellent discussion on insensible perspiration is recommended to the reader. Their conclusions are briefly summarized here. Two methods were employed for studying the insensible perspiration. With platform scales, sensitive to 10 Gm., the insensible loss was determined over

night during a period of nine or ten hours, thus enabling the calculation of the hourly loss with an accuracy of approximately 5 per cent. An extremely sensitive balance, capable of weighing 100 Kg. with an accuracy of 0.1 Gm., has also been used for measuring this loss in successive periods of from ten to fifteen minutes' duration. Measurements by these two methods indicate that in general the larger individual has the larger insensible perspiration. Refinement in the method of measurement brought out sharply the influence of body size, ingestion of food and exercise on the insensible loss. Since these factors likewise affect the metabolism, it is evident that there is a relationship between the insensible loss and the metabolism. Thyroid patients, whose metabolism is high, were found to have a large insensible loss, and diabetic patients, whose metabolism is low, were found to have a small loss. The relationship between the insensible perspiration and the metabolism of these two types of pathologic cases was such that when the data for the hourly insensible perspiration were plotted with reference to the twenty-four hour heat production, a straight line curve indicated the general trend of the metabolism. The ideal conditions for measuring the insensible perspiration so that the data may be used for comparative purposes require that the body should be warm and comfortable, and that the extremities, particularly in the case of women, should be well protected. This fact was brought out when it was noted that the environmental temperature, which profoundly affects the skin temperature, plays a decided rôle in the insensible loss. When the insensible perspiration of either pathologic patients or normal subjects was measured under these ideal conditions and the metabolism was determined at essentially the same time, it was demonstrated that the straight line curve based on the data for diabetic and thyroid patients may serve as an excellent index of the metabolism obtaining at the time of measurement of the insensible perspiration, even with normal subjects.

WATER METABOLISM. IV. SUGAR METABOLISM IN DEHYDRATION. E. ANDREWS, Arch. Int. Med. **38**:136 (July) 1926.

Sugar metabolism in dehydration is the fourth of a series of studies on water metabolism by Andrews. Sugar metabolism as illustrated by fall in blood sugar after administration of insulin is profoundly influenced by relative degrees of hydration of animals. The intensity and duration of fall in blood sugar after administration of insulin are enormously greater in animals who are dehydrated by various means and much less in animals who are flooded with water.

S. A. LEVINSON.

GASTRIC MOTILITY: THE MECHANISM OF THE PYLORUS. E. KLEIN, Arch. Surg. **12**:1224 (June) 1926.

Klein passes in critical review the various theories of an experimental work on the mechanism of closure and opening of the pylorus beginning with Cannon's theory of "acid control." From the work of others he concludes that this theory is inadequate, and from his own observations as well as those of many other workers, favors the mechanical theory as being most adequate and that fluidity of the stomach contents is a prime factor. His own experiments were those of direct observation on the stomach of dogs after food and after instillation of acid. These have been described previously. The pyloric ring is always in a state of tone, which is overcome by each peristaltic wave, with which the stomach contents are constantly being forced through to the

duodenum regardless of whether the reaction is acid or alkaline. The pylorus contracts firmly only when the wave reaches it, when it is effectively closed. He observed that solid bodies are forced back into the body of the stomach until reduced to a fluid state. Solid particles may be carried over at the end of digestion when the pyloric tone is lowest.

N. ENZER.

STUDIES ON THE INORGANIC COMPOSITION OF THE BLOOD. I. THE EFFECT OF HEMORRHAGE ON THE INORGANIC COMPOSITION OF SERUM AND CORPUSCLES. S. E. KERR, J. Biol. Chem. **67**:689, 1926. II. CHANGES IN THE POTASSIUM CONTENT OF ERYTHROCYTES UNDER CERTAIN EXPERIMENTAL CONDITIONS. Ibid. **67**:721, 1926.

A single hemorrhage of from one third to one half of the total blood volume results in a slight decrease in the sodium and chloride contents of the serum. A greater decrease, together with an increase in inorganic phosphate, follows repeated severe hemorrhage. No change occurs in the potassium, calcium or magnesium content of the serum, but there is a large increase in the potassium content of the corpuscles.

The potassium content of the corpuscles is increased following insulin overdosage or subcutaneous injection of sodium oxalate. The increase is accounted for as an actual penetration of the potassium into the erythrocyte where it is stored in a nondiffusible form.

ARTHUR LOCKE.

THE QUANTITATIVE ESTIMATION OF CALCIUM, MAGNESIUM, PHOSPHATE AND CARBONATE IN BONE. B. KRAMER and J. HOWLAND, J. Biol. Chem. **68**:711, 1926.

Methods are described in detail for the quantitative determination of calcium, magnesium, inorganic phosphorus and carbonate in from 0.5 to 1 mg. of bone.

STUDIES UPON THE INORGANIC COMPOSITION OF BONES. J. HOWLAND, W. M. MARRIOTT and B. KRAMER, J. Biol. Chem. **68**:721, 1926.

Different bones of the same individual have the same inorganic composition. The ratio of calcium phosphate to calcium carbonate is greater in the normal than in the rachitic animal. This ratio varies directly with the phosphorus content of the serum.

ARTHUR LOCKE.

SOME THOUGHTS AND EXPERIMENTS IN RELATION TO THE HORMONES: THE CRYSTALLIZATION OF INSULIN. JOHN J. ABEL, Proc. Inst. Med., Chicago **6**:108, 1926. CRYSTALLINE INSULIN. JOHN J. ABEL, Proc. Nat. Acad. Sc. **12**:132, 1926.

Commercial insulin contains 80 per cent or more noninsulin substance. According to a previous report (Researches in Insulin: I. Is Insulin an Unstable Sulphur Compound? J. J. Abel and E. M. K. Geiling, *J. Pharmacol. & Exper. Therap.* **25**:423, 1925), repeated precipitation with six normal pyridine and redissolving with sixth normal acetic acid; grinding of the precipitate with phenol (about 90 per cent.); recovering the active part from the phenolic supernatant with ether, alcohol or water, precipitating and finally separating the insoluble portion, leaves the acetic-acid-soluble, comparatively pure insulin "fraction 4." From a solution of acetic acid of this fraction, the contaminating

substance was precipitated with brucine (6 Gm. in 95 cc. sixth normal acetic acid) and the insulin precipitated by sixth normal pyridine. The crystals thus formed may be washed with distilled water, redissolved in acetic acid and reprecipitated as before; or they may be dissolved in fifteenth molar disodium hydrogen phosphate and then acidulated drop by drop with sixth normal acetic acid and left standing for slower precipitation which yields larger crystals. Insulin crystals are doubly refractive, of the rhombohedral division of the hexagonal system, melt sharply at 233 C., give a biuret reaction and positive Millon's, Pauly and ninhydrin tests, are soluble in dilute alkalis and acids but not in distilled water, and are easily rendered inert by boiling in tenth normal sodium carbonate (or sulphuric acid) which liberates the labile sulphur which seems to parallel quantitatively the hypoglycemic potency of the extract. As little as 0.01 mg. per kilogram of this purified insulin lowers the blood sugar of a rabbit to approximately the convulsive level of 0.045 per cent.

ETHEL B. PERRY.

PATHOLOGIC VARIATIONS IN THE SERUM CALCIUM. G. H. PERCIVAL and C. P. STEWART, Quart. J. Med. **75**:235, 1926.

Applying the method of Kramer and Tisdall, the authors find the normal range of blood calcium to be from 9.4 to 9.9 mg. per hundred cubic centimeters of serum. Parathyroid extract does not always raise this level, is occasionally without effect and may even lower it. Its action is sometimes augmented by the addition of calcium salts. Given alone, parathyroid extract did not raise the serum calcium in a case of tetania parathyropriva. The addition of calcium chloride caused a considerable rise. In three cases of lupus erythematosus the blood serum calcium was subnormal. In the presence of ketosis it was also found to be subnormal.

N. ENZER.

INFECTION OF THE URINARY TRACT IN ABSENCE OF VITAMIN A. GINO FRONTALI, Riv. di clin. pediat. **24**:505, 1926.

Each of seven white rats, which were fed a diet containing no vitamin A, developed infections of the urinary tract (abscess of the kidney, pyelitis, ureteritis, cystitis); nineteen control animals which were fed either the same diet plus vitamin A, or a normal diet, did not develop these infections. *B. coli* was found in seven cases: *Staphylococcus aureus* was also present in two cases.

Urinary infections are not unusual in artificially fed infants having nutritional disorders; the author therefore suggests that pyelocystitis in infants be treated by feeding vitamins.

HIGGINS, Cincinnati.

ACIDOPHILS. KARL SCHLIRF, Centralbl. f. Bakteriol. O., I **97**:104, 1926.

To call all the organisms isolated from the mouth, caries, vagina, milk, etc., "acidophils" is not correct. Schlirf terms them "acidotolerant." An acid medium is not necessary for their cultivation, and not always correct. He used a liver-liver-broth medium for isolation. He classified them as *Acidobacterium laetis, aerogenes, moroi, doederleinii* and *bulgaricum.* For their classification, the morphology, especially "width of the rods," is important, as well as further form on agar, gelatin, reaction of milk, gas or no gas production and degree of acidity in sugar medium.

E. E. ECKER.

THE ESTIMATION OF LACTOSE IN SMALL QUANTITIES OF BREAST MILK. ALEXANDER WITTENBERG, Monatschr. f. Kinderh. **32**:510, 1926.

The author devised a technic for the determination of lactose in breast milk, based on the principle of the Bang technic for the determination of sugar in the blood. With this method, he was able to show that the amount of lactose is not constant during the nursing. Like fat, it is less at the beginning and increases during the progress of the nursing. The variation is as much as from 1 to 2 per cent.

GERSTLEY, Chicago.

THE RELIABILITY OF BUERKER'S COLORIMETER, WITH SPECIAL REFERENCE TO THE HEMOGLOBIN DETERMINATION. F. LEBERMANN, München. med. Wchnschr. **72**:982 (June 12) 1925.

Buerker's colorimeter may be used for the quantitative determination of hemoglobin, salicylic acid, potassium cyanide, potassium bichromate, copper, copper sulphate and quinine. Using this and other methods, the author made comparative determinations and found that with Buerker's colorimeter smaller amounts of the unknown can be measured and with greater accuracy, than with Dubosq's or other colorimeters.

The results obtained using Buerker's colorimeter parallel those obtained with Sahli and Sahli-Leitz hemoglobinometer; with Buerker's instrument, a finer gradation is possible.

GROB, Detroit.

VAGINAL FLORA IN CHILDREN AND IN GIRLS AT THE AGE OF PUBERTY. G. SOEKEN, Ztschr. f. Kinderh. **40**:727, 1926.

The vaginal flora in children from earliest infancy on is represented chiefly by cocci. After the eleventh year, there is a change to bacilli in a majority of cases. The occurrence of a bacillary flora is connected with the coming of puberty, and one without the other was not observed in children beyond the first half year of life. The change of flora happens in an early stage of puberty, often a long time before the first menstruation and within a few days; in the cases observed, this change occurred within a period of eight days.

EMERSON, Boston.

Nutrition

PRODUCTION OF VITAMIN BY BACTERIA. W. KOLLATH and B. LEICHTENTRITT, Centralbl. f. Bakteriol. O., I **97**:119, 1926.

It was impossible to prevent scurvy in the guinea-pig by feeding the animals *B. coli*, mixture of bacilli from the intestines of the guinea-pig, or Friedländer's bacillus (living or killed at 56 C.) or by medium itself. Experiments therefore demonstrate that the growth stimulating factors for influenza bacilli are not the same as the real vitamins.

E. E. ECKER.

IS THE ACTIVITY OF SUNLIGHT IN RICKETS DEPENDENT ON THE COINCIDENT ADDITION OF VITAMINE? H. VOLLMER, Ztschr. f. Kinderh. **40**:655, 1926.

A pair of twins, 26 months old, had acquired severe rickets in spite of an early and generous diet of vegetables and fruit, and in spite of the administration of cod liver oil for a year. One could not determine whether or not the vitamin afforded insufficient protection or whether there was an insufficient

amount of vitamin. In the month of March, by intensive eosin and sun treatment, even with a diet extremely poor in vitamin, the author succeeded in paving the way for a cure, and obtained definite laying down of calcium in the bones in fourteen days. Generous addition later of vitamin, all factors, did not favor the progress of this treatment. A comparison of the time required for treatment with vitamin exclusively and with light exposure exclusively shows the overwhelming superiority of light.

INFLUENCE OF CORRELATION OF NUTRITIVE SUBSTANCE ON THE ABSORPTION OF SUGAR. E. NASSAU and S. SCHAFERSTEIN, Ztschr. f. Kinderh. **40**:659, 1926.

The significance of individual food elements for the digestive processes in the intestine and for the pathogenesis of disturbance of nutrition cannot be determined by following out the fate of any single substance. Only the quantitative relation of individual substances to one another determines what happens in the intestine and in the metabolism of healthy and of sick babies. The long stay or the quick absorption of sugar in the stomach and intestines depends exclusively on the quality of the nutritive substances offered at the same time. A pure solution of sugar is quickly absorbed from the intestines. The addition of albumin, and still more that of fat and even more mixtures which contain cow's whey, delays sugar absorption. Human whey, in comparison, delays it only a little. This supports the view of Finkelstein and others that diarrhea does not occur after the feeding of pure sugar solution. The quick disappearance of sugar from the intestine avoids bacterial fermentation. Mixtures holding back the sugar in the intestine favor fermentation.

THE DETERMINATION OF THE CALCIUM CONTENT OF HUMAN MILK. E. STRANSKY, Ztschr. f. Kinderh. **40**:671, 1926.

The method of Kramer and Tisdall for determining the calcium content of blood lent itself very well to the determination of calcium in human milk. Only small amounts, 2 cc., are needed. Human milk before the baby is nursed shows a higher calcium content than during the nursing or afterward.

ANTIRACHITIC ACTIVATION, BY EXPOSURE TO LIGHT, OF DIETS PRODUCING RICKETS. I. SEREBRIJSKI, H. VOLLMER and E. ZADECK, Ztschr. f. Kinderh. **40**:716, 1926.

The authors state that the astonishing work of several American investigators on the power of ultraviolet rays to confer antirachitic properties on inactive nutritive substances was received in Germany with great doubt, but experiments there were confirmatory. The Sherman-Pappenheimer diet and also the McCollum, Simmonds, Shipley and Park diet, both productive of rickets, acquired antirachitic activity; they protected the experimental animals more or less completely from rickets but were not able to prevent xerophthalmia. One must assume that the treated foods influenced the animals in a way which might be characterized by saying that they increased the need of a factor without covering it. No growth-producing activity was found.

EMERSON, Boston.

STUDIES IN THE VITAMIN B CONTENT (RAT-GROWTH-PROMOTING WATER-SOLUBLE B) IN THE MOTHER'S MILK OF INFANTS SUFFERING FROM INFANT BERIBERI. TADASHI SUZUKI, Oriental J. Dis. Inf. **1**:51, 1926.

Beriberi is more common in infants than in adults, the vitamin not being so necessary for the adults. Breast-fed children may develop beriberi.

The author experimented with white rats which received a diet containing no vitamin B. When less than 4 cc. of milk from mothers having children with beriberi was added to this diet, the rats showed signs of "avitaminosis"; 4 cc. of this milk protected the rats. Milk from mothers of normal children was protective to the rats in a quantity as small as 1 cc. A commercial vitamin B product (oryzanin) was also successful in protecting rats.

Beriberi in infants and "avitaminosis" in rats do not show precisely the same symptoms. Thus, although milk from mothers with beriberi is deficient in vitamin B, it is not proved that beriberi is caused by lack of vitamin B.

HIGGINS, Cincinnati.

Prenatal Conditions

SOLE PATTERNS OF TWINS. R. B. MONTGOMERY, Biol. Bull. **50**:293 (April) 1926.

"In the study of twins, one frequently desires to know whether or not they are of monozygotic or dizygotic origin." In the new-born, examination of the placenta and membranes answers this problem, but in children and adults other methods are necessary. In such cases the study of the general physical appearance, mentality, palm patterns, whorls of the hair, etc., have led to no definite results, and Montgomery undertook the study of sole patterns in the hope of revealing some means of identification.

Prints were secured from eighty-seven pairs of twins by a process perfected by Mathews. The age of the youngest pair was 1 week, and of the oldest, 85 years. Most of the subjects, however, were school children. Of the eighty-seven pairs of twins, thirty-three pairs were male-male, twenty-four were female-female and thirty were male-female. Newman thinks that monozygotic twins are likely to have identical patterns, but that unlike patterns do not disprove monozygotic origin. Wilder also has remarked on the striking similarity which sometimes occurs but declares that, while they are similar grossly, the Galton details are always different. Montgomery found thirteen sets in his series of eighty-seven pairs of twins with identical patterns, or a percentage of 14.94. In all except one pair, this similarity of pattern occurred in like sex twins. He concludes that "the presence of identical patterns on the soles of a pair of twins might point to their monozygotic origin, but as Newman states, their absence does not disprove it."

ALPERS, Philadelphia.

THE ROENTGEN-RAY IRRADIATION OF THE EGG AND EMBRYO. CULTURES IN VITRO. ANGEL H. ROFFO, Bol. Inst. med. Exper. **3**:143, 1926.

Cultures prepared from embryonic chick tissues soon after irradiation within the egg, even with large doses, developed as normal tissues; but if forty-eight hours intervened between irradiation and culturing, or even twenty-four hours if the embryo had been separated from the egg, the tissue was already in a state of disintegration, and development either failed or was weak. This behavior suggests that physicochemical and hormonic changes of the medium may result from the roentgen-ray treatment.

[ARCH. PATH.]

EXPERIMENTAL STUDY ON TOXICITY OF EMBRYONIC EXTRACTS OF THE CHICKEN AND THEIR ACTION ON BLOOD PRESSURE. A. H. ROFFO and R. LOPEZ RAMIREZ, Bol. Inst. med. Exper. **3**:211, 1926.

Fresh extracts of chick embryos in physiologic salt solution (dilution, 30 per cent) are found to be toxic for adult fowls only during the last days of

incubation; even those of from five to seven days are little effective. Hypotension and blood coagulation are produced. Elimination of the albumin by heating or filtration of the extracts makes them innocuous.

ETHEL B. PERRY.

Acute Infections

INTRADERMAL VACCINATION AGAINST SMALLPOX. M. BUSSEL and R. STANKIEWICZ, Rev. franç. de Pédiat. **2**:293, 1926.

In addition to a review of the literature, the authors describe their experience with 683 patients in whom intradermal vaccination had been used. They conclude that excellent results are obtained with a minimum of risk if the vaccine is sufficiently diluted, 1:80 or 1:100. In ordinary subjects this method avoids the dangers of secondary infection and inoculation of other parts of the body with the vaccine. In patients with eczema, an exudative diathesis or any chronic skin condition, it is considered invaluable.

LESLIE, Chicago.

EXPERIMENTAL SCARLET FEVER. S. J. ZLATOGOROFF, W. S. DERKATSCH and S. J. NASLEDYSCHEWA, Centralbl. f. Bakteriol. O., I **97**:152, 1926.

Rabbits and monkeys (*Macacus rhesus*) do not show in experimental scarlet fever the same picture as occurs in man. Experimental scarlet fever is a disease "sui generis." Experimental scarlet fever in animals can be produced with materials containing streptococci (blood, organs, etc.) or with materials free from the organisms. The substance producing the disease passes through a filter, and is found in the mouth, blood and organs of the patients. A "passive virus" can be obtained from animals. It activates the streptococcus and confers on the organisms antigenic qualities.

E. E. ECKER.

DIPHTHERIA OF THE WOUND IN AURAL SURGERY. LEONARD SEIFERTH, Ztschr. f. Hals-, Nasen- u. Ohrenh. **14**:519 (June 12) 1926.

Seiferth reports two cases of his own and refers to others. The course of diphtheria of the wound can vary very much. In the great majority of cases the complication is harmless. At times, postdiphtheritic paralyses are observed (paralysis of the soft palate and paralysis of accommodation). Polyneuritis has also been recorded. Twenty-one patients have died since 1918. If the general condition of the patient is not particularly affected, and if no alarming symptoms occur (high fever, paralyses), the diphtheria of the wound must be regarded as a local condition, and the prognosis is favorable. If it shows the disease picture as it presents itself in a diphtheritic condition of the throat, it must be considered a general disease. In the first instance the ordinary antiseptics can be used in the wound, or antidiphtheritic dry serum can be sprinkled on it. The use of iodoglycerine, which was used with success in nasal diphtheria, proved to be successful in one of the author's cases when applied to the wound daily. In grave conditions, however, antidiphtheritic serum must be injected in addition to the use of local therapy. During the last few years, the variability of diphtheria bacilli has been investigated. Von Bernhardt and Paneth transformed diphtheria bacilli by cultivation; Jacobsthal cultivated various types from the blood of a cadaver. If investigation will solve this problem, more can be learned about the multiformity of diphtheria.

AMBERG, Detroit.

BODY TEMPERATURE IN MEASLES AT THE TIME OF THE INFECTION AND DURING INCUBATION. H. OREL, Ztschr. F. Kinderh. **40**:623, 1926.

Twenty-six cases are reported, in which an increase of temperature was looked for at various times from the beginning of measles to the end of the period of incubation, and it was found that in the majority of cases no increase occurred. When there was an increase of temperature it was not due to the measles virus, but probably to a mixed infection.

EMERSON, Boston.

Chronic Infections

LEPROSY IN A FOURTEEN MONTHS OLD CHILD. E. A. NEFF, J. Trop. Med. **29**:146 (May 15) 1926.

The patient in the case reported is stated to be the youngest on record, the history being that of a child born of an actively leprous mother. Fourteen months after birth the first nodule was noticed on the cheek and was positive for *Bacillus leprae*. Several small depigmented areas were also observed on the shoulder and back. After seven months' treatment with chaulmoogra oil the disease became stationary and the bacteriologic reaction negative for acid-fast organisms.

[ARCH. DERMAT. & SYPH.]

THE RELATION OF INFANTILE HEMIPLEGIA TO HEREDITARY SYPHILIS. L. BABONNEIX, Arch. de méd. d. enf. **29**:561 (Oct.) 1926.

Few questions have been more vigorously debated than this, not only in France, where the efforts of Fournier and his students have clarified so many disputed points in heredosyphilis, but elsewhere. While most writers assert that a definite relation exists between infantile hemiplegia and inherited syphilis, they are still many distinguished clinicians, among whom may be mentioned Foerster, Freud, König, Young and B. Sachs, who oppose this contention, and feel that juvenile syphilis plays no particular rôle in the development of hemiplegia. Silverskold, an eminent Scandinavian authority, basing his conclusions on a study of ninety-one cases noted personally, favors the latter theory. From a careful scrutiny of thirty-eight cases, Babonneix found frank evidences of inherited syphilis in twelve. He quotes complete histories of these children, and concludes that it is as logical to select syphilis as a causative agent in hemiplegia of early infancy as it is to incriminate the same virus in tabes, general paralysis or leukoplakia buccalis of later life. The law of coincidence, while generally recognized as an interesting phenomenon, can hardly be called on to support a scientific thesis, and the author acknowledges the force of Freud's argument when he suggests that "syphilis of the parents and hereditary syphilis of the hemiplegic infant are two distinct things, and the coincidence of lues in the family history and cerebral paralysis cannot be invoked as proof without more ample discussion."

AMESSE, Denver.

PATHOGENESIS OF HEREDITARY SYPHILIS. G. PICCARDI, Gior. ital. di dermat. e sifii. **67**:22 (April) 1926.

The transmission of syphilis to the fetus may occur from both parents or from the mother alone. The paternal transmission of syphilis is doubtful. In fifty-eight personal observations of abortions, stillbirths and children born with clinical manifestations of syphilis, the mother was infected in every case. On

the other hand, the children of men with active syphilis of the skin, heart or nervous system were always born free from any symptoms when the mother had not been infected.

NEW SERUM DIAGNOSIS OF SYPHILIS. O. SCIARRA, Riforma med. **42**:533 (June 7) 1926.

The blood serum of syphilitic patients presents numerous ultramicroscopic granules, free or agglutinated, of different size and mobility. If absolute alcohol is added to the serum, these particles are dispersed rapidly and uniformly. These liberated particles will again react in the presence of complement, fixing it and showing its deviation when a hemolytic system is added. The technic of the reaction is as follows:

Inactivated Serum, Cc.	Alcohol, Cc.	Complement, Cc.	Incubation, at 37 C.	Hemolytic System, Cc.	Incubation, at 37 C.	Result	
						Negative	Positive
0.2-0.3	0.1-0.15	According to titration	45 to 60 minutes	1	1 to 2 hours	Hemolysis —	No hemolysis ++++

[ARCH. DERMAT. & SYPH.]

Internal Diseases

VALVULAR DISEASES OF THE HEART WITH SPECIAL REFERENCE TO THE PATHOGENESIS OF OLD VALVULAR DEFECTS. B. J. CLAWSON, E. T. BELL and T. B. HARTZELL, Am. J. Path. **2**:193, 1926.

In addition to the vegetations in acute endocarditis there is a diffuse inflammation always in the free edge and often involving the greater part of the leaflet. This circumstance explains the uniform thickening so commonly seen in old defective valves.

Rheumatic vegetations are composed chiefly of fibroblasts, and in the process of healing they readily become converted into fibrous tissue. There is no ulceration and no organization. Fifty-five of seventy-three old defective valves were considered the result of rheumatic endocarditis, and in twenty-seven of these, incompletely healed rheumatic lesions were recognizable.

Bacterial endocarditis is a more intense inflammation than the rheumatic form. Proliferation predominates, but exudation is often prominent. Large thrombi are formed on the raw surfaces, and there is often ulceration. Healing consists of the conversion of the leaflet into scar tissue. Such portions of the thrombi as do not become detached persist indefinitely without becoming organized, although they may become calcified. Complete healing rarely occurs. Three of seventy-three old defective valves were interpreted as the result of bacterial endocarditis.

Transitions between rheumatic and bacterial vegetations are frequently seen. Rheumatic vegetations were found in association with bacterial in three-fourths of the cases of subacute bacterial endocarditis.

Fifteen of seventy-three old defective valves were of the aortic calcified nodular group. The etiology of this type is unknown. There is no satisfactory evidence that it is of inflammatory origin, and it seems unrelated to atheroma. Aortic stenosis, in the absence of disease of any other valve, is usually of this form.

Stenosis is more frequent than insufficiency in old defective valves. The only pulmonary valve defects seen were of the congenital type (three cases of pulmonary stenosis).

An acute rheumatic endocarditis may terminate in several different ways: (*a*) death during the acute stage from toxemia; (*b*) partial or complete healing followed after a variable interval by the reappearance of fresh rheumatic vegetations (recurrent rheumatic endocarditis); (*c*) partial or complete healing followed by the formation of bacterial vegetations on the valves—a more active inflammation (subacute bacterial endocarditis); (*d*) slow incomplete healing giving rise to deformed leaflets on which rheumatic inflammation is still recognizable; (*e*) complete healing resulting in thickened, stiffened valves with smooth surfaces.

As to pathogenesis, old valvular defects in seventy-six hearts were interpreted as follows: fifty-five were caused by rheumatic endocarditis, three by bacterial endocarditis, fifteen (all aortic stenosis of the calcified nodular type) of undetermined origin and three (pulmonary stenosis) congenital.

AUTHORS' SUMMARY.

ACUTE PERICARDITIS. HERMAN ELWYN, M. J. & Rec. **124**:6 (July 7) 1926.

Acute pericarditis occurs in the course of rheumatic conditions as a manifestation which indicates the degree of severity with which the heart is affected. It also occurs in the course of other diseases. An inflammatory exudate in the pericardium modifies the function of the heart in a twofold manner.

1. The inflammation may involve the ventricle muscle, and the contractility of the ventricle is consequently impaired.

2. The exudate causes a mechanical embarrassment. The greater the amount of exudate, the greater the embarrassment. Normally, one of the functions of the pericardial sac is to prevent overdistention of the heart. Experiments are given to show the behavior of the pericardium when fluid is injected. The author then describes the clinical forms of acute pericarditis, giving the clinical features in the various forms. Acute pericarditis occurs most frequently in the course of rheumatic fever. At times the primary disease which is responsible for the presence of an acute pericarditis is not at all obvious. In these cases there are some considerations which may help to determine what the primary disease is. For instance, in young people a rheumatic condition of the heart is the chief causative factor, while in patients past middle age coronary obstruction followed by myocardial infarction is the chief cause. Treatment should be directed (1) to the primary disease; (2) toward lessening the work of the heart, and (3) toward alleviating any annoying symptom. The treatment of purulent pericarditis is surgical.

STATUS LYMPHATICUS. R. S. LEADINGHAM, M. J. & Rec. **124**:345 (Sept. 15) 1926.

The author discusses the embryology and physiology of the thymus gland, and gives a clinical picture of status lymphaticus. He then discusses the cause of death from this disease and draws the following conclusions: (1) We do not know the cause of status lymphaticus. (2) We look to the laboratory for further information concerning the interrelationship of lymphoid and thymic development with the secretions of ductless glands. (3) We should secure postmortem examinations in all cases of sudden death.

WILLIAMSON, New Orleans.

ACRODYNIA IN THE UNITED STATES. W. WESTON, South. M. J. **19**:665, 1926.

Regarding the etiology of this condition, practically only three hypotheses are now considered. It may be neuritis following a respiratory infection; it may be the result of a focal infection, or it may be a food deficiency disease. The author classes acrodynia among the food deficiency diseases, and believes that the condition described by French physicians as early as 1828 is identical with the condition described today which is considered a new disease manifestation. He would retain the same acrodynia, first applied to the condition by the French physician, Chardon.

ALIMENTARY ANEMIA IN INFANCY. CARROLL M. POUNDERS, South. M. J. **19**:727, 1926.

Premature infants, those born of anemic mothers and those fed on a high carbohydrate diet or on an exclusive milk diet during the first fifteen months often show a marked secondary anemia. The cause may be an insufficient amount of iron stored up during the last months of intra-uterine life, or an insufficient intake of iron in the food. There is a deficiency of hemoglobin out of proportion to the reduction in the erythrocyte count.

In multiple pregnancy or pregnancy in an anemic woman vigorous treatment should be instituted during the latter half of pregnancy in an effort to increase the amount of iron reserve in the body of the infant. The author also regards ultraviolet ray therapy as a valuable agent.

SCHLUTZ, Minneapolis.

THE FUNCTIONAL PATHOLOGY OF NEPHRITIS. E. B. MAYAS, Quart. J. Med. **75**:273, 1926.

On the basis that the kidney exercises two functions, filtration and concentration, Mayas accepts MacLean's classification of nephritis into hydremic and azotemic types. The former, primarily a disturbance of filtration, may develop hyposthenuria. In support of the theory that the chief cause of nephritic edema is failure of the plasma proteins to retain water in the circulation, Mayas devised a method of measuring the colloid osmotic pressure of the plasma proteins, which is exerted chiefly by serum albumin. He found that considerable changes in the salt content of the plasma had practically no effect on the colloid osmotic pressure, which, of course, does not support the view that chloride retention is of importance in the causation of edema. In hydremic nephritis the colloid osmotic pressure is reduced to a fourth of the normal value. The plasma in these cases contains less protein than normal serum, but the osmotic pressure is even lower than normal serum diluted to an equal protein content. In cardiac failure the edema occurs at a higher level of colloid osmotic pressure and appears to be due to an increase in capillary blood pressure from venous stasis and possibly from disturbance in nutrition of capillary endothelium as well. The depression of the freezing point and the concentrations of urea and chloride were determined in the plasma and urine of a number of patients with azotemic (chronic interstitial) nephritis. From his observations Mayas concludes that there is no evidence of lessened permeability of the glomerular membrane to dissolved substances, but it is impossible to determine whether a sufficient number remain active to produce a normal amount of filtrate. The tubules are unable to concentrate the filtrate, although they are able in most cases to reabsorb the chlorides. The failure to

concentrate the glomerular filtrate is due to a lowered osmotic resistance of the tubule cells to urea, allowing it to diffuse passively through them.

N. Enzer.

Evolutive Cardiac Rheumatism with Multiple Pulmonary Infarction and Specific Nodular Myocarditis. Edouard Pichon and Carlos R. Larde-Arthes, Arch. de méd. d. enf. **29**:581 (Oct.) 1926.

Few instances of nodular myocarditis following rheumatism have been published in France. Letulle, collaborating with Bezancon and Weil, reported a case during the current year in which the Aschoff bodies were demonstrated. The author's case is described in great detail, the salient features being as follows: A boy, aged 14, enjoying excellent health throughout infancy and childhood, was seized in August, 1925, with a violent attack of rheumatism, involving the right elbow and the major joints of the lower limbs. This confined him to bed for two weeks, when he was able to return to work, but with considerable stiffness remaining in the legs. Toward the end of September a second attack occurred, the boy complaining of severe pains in the right hypochondrium. Examination at this time revealed a gallop rhythm with dilatation of the heart and distention of the abdomen from passive congestion of the liver. Suitable treatment again permitted him to return to work, but with a badly injured heart. On Feb. 5, 1926, he suffered a complete collapse, and after ten days' nursing at home he was sent to the Hospital for Sick Children (Paris). Here it was noted that, in addition to the cyanosis, the boy had a morbilliform eruption involving the chest and upper limbs. Pancarditis, with the usual clinical phenomena, was present. In spite of intensive treatment with digitalis and salicylates, pulmonary infarction of the right lung developed on February 27, the patient expectorating blood freely. His condition gradually grew worse until the day of decease, March 15. Necropsy revealed the lungs to be edematous and strewn with infarcts of various size, the two largest being situated, respectively, in the inferior portion of the upper lobe of the right lung and in the superior part of the lower lobe of the left lung. They were irregular in contour; in fact, none of these infarctions showed the usual pyramidal form. Thrombosis could not be found in the lung tissue. The pericardial sac was filled with a serosanguineous fluid, and the surface of the heart was covered with a fibrinous exudate, which was particularly abundant on the anterior aspect. Examination of the heart showed tricuspid insufficiency without endocarditis; the mitral valves were softened at the borders and were occupied by pearl-like rose colored elevations about the size of a millet seed. The aortic valves also showed lesions of rheumatic endocarditis. Microscopic study of the myocardium revealed fragmentation of the muscle fibers, and the nodules of Aschoff were readily demonstrated in the fibrous tissue as well as between the muscle bundles. The origin and nature of these bodies are freely discussed and the literature abstracted.

Amesse, Denver.

The Question of Chronic Albuminuria in Children. E. Faerber and Telemachos Demetriades, Jahrb. f. Kinderh. **113**:119-127 (July) 1926.

Acute nephritis in children usually heals after a certain length of time, and the question of chronic nephritis need not be considered. There are children in whose urine albumin, cylindroids and morphologic elements occur over a

long period. Heubner classifies these conditions as pedonephritis, and considers that they have nothing to do with chronic nephritis as in the adult. He considers treatment of this condition unnecessary, for the associated symptoms of chronic nephritis never develop. He considers the cases as distinct from orthostatic albuminuria also, because in one such case degenerative changes were found in the kidneys at necropsy. Casts are more frequently found in the urine, and often the kidneys do not heal for many years.

Jehle, on the other hand, showed that in orthostatic albuminuria cylindroids, red and white blood cells and epithelial cells may occur in large numbers and may persist even with rest in bed, so that they cannot be distinguished from pedonephritis. He considers these only as a special form of orthostatic albuminuria which occurs in children whose kidneys are so sensitive that even with rest in bed slight changes in the position of the body are capable of producing albuminuria, cylindruria and hematuria. Four cases are cited in which albumin and morphologic forms were present in the urine even after rest in bed, so that the specimen of urine in the morning was not free from albumin. Two of these patients had a preceding history of scarlet fever. Tests showed the presence of albumin precipitated by cold acetic acid, and also serum albumin as shown by the acetic acid-potassium ferrocyanide test. Even the albumin precipitated by cold acetic acid may disappear during forced lordosis, and the serum albumin alone remains, giving the picture of true chronic nephritis. When a kyphotic position was assumed, all sediment disappeared from all specimens in a few hours (about three), but recurred again when a lordotic position was assumed.

EHRMANN, Chicago.

THERAPY OF CYSTOPYELITIS. E. TRENDTEL, Klin. Wchnschr. **5**:1275, 1926.

Trendtel reports the successful treatment of patients who had both acute and chronic cystopyelitis with bladder irrigations of 0.25 to 1 per cent silver nitrate. The use of stronger solutions is dangerous. From four to five irrigations at three day intervals are given. The bladder is first washed out with a warm 3 per cent boric acid solution, then from 50 to 100 cc. of the silver nitrate solution is allowed to flow in. This is followed by 1 per cent sodium chloride solution.

SCHWARTZ, Milwaukee.

THE CONSTITUTIONAL PATHOLOGY OF CONGENITAL PYLORIC STENOSIS. W. LASCH, München. med. Wchnschr. **72**:1155 (July 10) 1925.

The author gives the history of monochorionic twins. One of the twins developed symptoms of pyloric stenosis and died in the course of the disease. The postmortem examination revealed a typical pyloric tumor. The other twin developed normally without having any signs of pyloric stenosis.

GROB, Detroit.

THE RÔLE OF BACTERIA IN THE ETIOLOGY OF BRONCHIAL ASTHMA AND THE RELATIONSHIP BETWEEN ANAPHYLAXIS AND IMMUNITY TO TUBERCULOSIS IN THE HUMAN SUBJECT. H. GROSSFELD, Wien. Arch. f. inn. Med. **13**:117, 1926.

From an investigation of 215 cases of bronchial asthma, the author concludes that most true cases of bronchial asthma are of tuberculous origin. He conceives tuberculous bronchial asthma as an anaphylactic reaction between

bacterial antigen and the free circulating antibodies developed from the protein of the tubercle bacillus.

Experimental studies have shown that in the true asthmatic attack there is a venous stasis and hyperemia in the bronchial vessels. This hyperemia is possibly a defensive reaction against tuberculosis, and may explain the peculiar antagonism which exists between tuberculosis and asthma as expressed in the apparent immunity of the asthmatic subject to this particular infection.

The heterogeneous factors which precipitate an attack are generally nonspecific external or internal irritants, which in the tuberculous subject develop endogenous antigen production. This in turn precipitates the anaphylactic reaction.

SCHLUTZ, Minneapolis.

FAMILIAL ICTERUS OCCURRING WITH CIRRHOSIS OF THE LIVER AT PUBERTY. H. BISCHOFF and R. BRÜHL, Ztschr. f. Kinderh. **40**:702, 1926.

Three sisters at the age of 11 or 12, in succession, became ill with chronic jaundice and considerable enlargement of the liver and spleen. Development of the girls stopped at the first symptom. The disease continued with remissions, and the older the girls became, the more severe became the acute attacks, in one of which the eldest girl finally died at the age of 16. The normal resistance of the red blood corpuscles to hypotonic salt solution spoke against familial hemolytic anemia, as did also the itching of the skin and acholic stools. Against the diagnosis of familial splenic anemia was the severe jaundice, ascites and the itching skin. It could not have been dermatitis exfoliativa because there was no involvement of the nervous system. The fact that the disease occurred in three sisters at puberty, while two brothers were healthy, and the fact that there was manifest general physical retardation allow the authors to assume a constitutional disturbance connected with the endocrine system. At necropsy, the eldest girl showed two processes going on: cirrhosis of the liver and infantilism. This picture corresponds to no known disease, and is thought to be due to a toxin formed, perhaps in the intestine, especially in a body already suffering from a condition similar to infantilism.

EMERSON, Boston.

THE DIAGNOSIS OF BRONCHIECTASIS BY LIPIODOL. J. SVEJCAR and F. DREUSCHUH, Čas. lék. česk. **64**:568 (April 11) 1925.

After a review of the literature, the authors present a report of two cases. In one of the patients the instillation had to be performed by bronchoscopy, because of an anomaly of the thyroid, so that the usual tracheal puncture could not be made. The results of extended study may throw some light on the etiology, especially since there is a question of congenital syphilis, congenital defect and posttraumatic and postinfectious elements. This method is of value in differentiating cavitation. The authors feel that a great many cases of bronchiectasis will be uncovered by an extended use of iodized oil and by roentgen-ray study.

LEGG-CALVE-PERTHES SYNDROME; MALUM COXAE JUVENILE, A DISCUSSION OF ITS ETIOLOGY AND PATHOGENESIS. RICHARD RIPA and VLADIMIR MUSIL, Čas. lék. česk. **64**:644 (April) 1925.

This is a review of the literature on diseases of the hip joint which do not fit into the category of congenital, syphilitic, rachitic, tuberculous or arthritis

deformans. Trauma is cited as a possible etiologic factor. Little pain and a limp for a year or more comprise the usual initial history. Clinical observations antedate the roentgen-ray observation of epiphyseal rarefaction and diminution in the size of the head of the femur. Schwarz reported forty-four cases in boys and eleven in girls; Waldenstom reported five cases in girls and thirty-five in boys; Broca reported the condition in one girl and in ten boys and Ripa and Musil report the condition in four girls and in twelve boys. The age incidence is from 5 to 10 years. The summary of therapeusis is a limitation of activity for a period of from four to five years.

STULIK, Chicago.

INVESTIGATION OF THE DURATION OF "NEW GLUCOSE" OCCURRENCE IN THE BLOOD OF DIABETIC PATIENTS AFTER A SINGLE INJECTION OF INSULIN. EINAR RUD, Ugesk. f. Læger **33**:760, 1926.

The work of Rud, in numbers 32 and 33 of *Ugeskrift for Læger,* tends to corroborate the observations of Lundsgaard and Holbøll in regard to the blood sugar designated as "new-glucose." This sugar occurs in the blood of a diabetic subject from one to two hours after a single injection of insulin, and disappears in the course of three or four hours. The author points out a significant relationship between the time of rise of the respiratory quotient, as Holten shows occurs from two to four hours after an injection of insulin, and the appearance of "new glucose" in the blood.

It is still a question in the minds of many investigators whether this new form of glucose actually occurs in the blood after the use of insulin. Several American investigators have been unable to obtain the changes reported by Lundsgaard and Holbøll.

SCHLUTZ, Minneapois.

Nervous Diseases

STUDIES IN SCHOOL CHILDREN FROM A NEUROPSYCHIATRIC VIEWPOINT. G. L. ECHOLS, J. M. A. Georgia **15**:303, 1926.

This interesting paper is a plea for a closer study of the child who does not progress normally in school. Five cases are reported in order to bring out the importance of this work.

MASHBURN, Atlanta.

CONVULSIONS IN INFANCY AND CHILDHOOD. M. G. PETERMAN, Wisconsin M. J. **25**:181, 1926.

Convulsions of infancy and childhood are classified as being due to organic, functional and acute infectious diseases. A convulsion in a new-born infant is probably organic, and cerebral hemorrhage, birth injury and congenital malformations of the brain should be considered in their respective order. A spinal puncture should be made. Convulsions occurring within the first few days of life and at 8 months are usually due to congenital malformations of the brain or skull and acute infections. Most of the convulsions occurring between the ages of 8 months and 3 years are functional, due to spasmophilia. Acute infections, including encephalitis, are next in frequency and are usually accompanied by an obvious history. Epilepsy, brain tumor and congenital malformations must always be kept in mind, more especially in older children from 3 to 15 years of age. Recurring convulsions, absence of fever and general symptoms are suggestive.

KELLY, Milwaukee.

THE INHERITANCE OF EPILEPSY. W. BRAIN, Quart. J. Med. **75**:299, 1926.

In a series of 200 epileptic patients, 28 per cent gave a family history of the disease as compared with less than 10 per cent in a control series from a hospital population. In those with a family history the onset was earlier, and the family history was present more frequently in females. The author considers that this indicates that there is an inherited predisposition to the disease.

THE SPASM OF TETANY CONSIDERED AS A DISTURBANCE OF THE PHYSIOLOGY OF MUSCLE. J. P. MARTIN, Quart. J. Med. **75**:311, 1926.

The spasm of tetany is similar to that of myotonia. In tetany there is a reduction in blood calcium, and in many cases an alkalosis is present. Lactic acid and phosphoric acid are the immediate activators of muscle contraction, and these are derived from a precursor, lactacidogen. Calcium and reduced hydrogen ion concentration inhibit the production of these acids and they therefore will be produced more readily when the blood calcium is lowered and in the presence of alkalosis. He therefore considers that the spasm of tetany is due to a disturbance in the muscle metabolism and is not neurogenous.

N. ENZER.

A CASE OF CHRONIC ANTERIOR POLIOMYELITIS TRANSITORY TO LATERAL AMYOTROPHIC SCLEROSIS. A. FOIX and J. A. CHARANY, J. de neurol. et de psychiat., February, 1926.

The authors describe a case of chronic poliomyelitis which during the life of the patient was accompanied by signs of pyramidal tract involvement (tendency to clonus and to the Babinski sign). These signs caused a suspicion of amyotrophic lateral sclerosis. The diagnosis was, however, discarded because of the unusually long course of the disease. From the pathologic point of view, the main aspects were the lesions of both anterior horns of the spinal cord. The nerve cells were largely involved and atrophied, especially in the cervicodorsal region. Besides the lesions of the nerve cells, there was an evident degeneration of the pyramidal tract at the level of the sacral region. The involvement of the pyramidal tract was moderate and localized.

The question arises whether we are dealing here with a stationary lesion or with one that is extremely slow in evolution and the result of which becomes manifest late. The fact that the slight pyramidal signs had been present since the early stages of the disease makes the authors incline toward the first hypothesis. They emphasize this combination of lesions of typical chronic poliomyelitis with moderate localized lesions of the pyramidal tracts in order to raise the question of the transitional forms between chronic poliomyelitis and amyotrophic lateral sclerosis. According to their point of view, no barrier exists between the two diseases.

FERRARO, Washington, D. C.

THE BLOOD p_H IN SPASMOPHILIA. P. ROHMER and P. WORINGER, Rev. franç. de Pédiat. **2**:319, 1926.

No significant variation from the normal was found in the p_H of the blood of infants with latent or manifest spasmophilia. The alkaline reserve may be normal, but is often lowered.

LESLIE, Chicago.

ENKOPRESIS. S. WEISSENBERG, Ztschr. f. Kinderh. **40**:671, 1926.

Enkopresis, or involuntary defecation, as compared to enuresis is much less frequent, 0.5 per cent as compared to 10 per cent. It is a habit of short duration which does not occur as regularly as enuresis, and occurs in younger children. As might be expected, every case of enkopresis is affected by enuresis. The rôle of dreams, as in bed-wetting, and emotions, such as anxiety, are deciding factors.

CLINICAL SEQUELAE AND THEIR PATHOGONESIS IN EPIDEMIC ENCEPHALITIS IN CHILDREN. L. KWINT, Ztschr. f. Kinderh. **40**:678, 1926.

Forty-two children of preschool and of school age in the chronic stage of encephalitis were studied clinically and psychologically under epidemic conditions in Russia.

1. The parkinsonian syndrome, which occurred in twelve cases, was observed much more rarely than in adults and much more frequently in older than in younger children.

2. Characteristic of the chronic form of encephalitis is the peculiar disturbance of sleep and the change in personality and behavior. In the majority of cases, the disturbance in sleep consisted of nocturnal sleeplessness with peculiar psychomotor unrest and the turning of night time into day. Drowsiness in the day, in the absence of nocturnal disturbance of sleep, occurred much less often.

3. Changes in personality and behavior represent a particular subcortical psychopathic syndrome which expresses itself in a deep disturbance of character, or incompatibility, in antisocial tendencies such as lying and stealing, and in sexual aggressiveness. These children know the difference between right and wrong but cannot withstand an imperative impulse. Behavior is injured more than character. The disturbance of personality was without any demonstrable abnormality of the nervous system pathognomonic of encephalitis; it occurred without any change in conditions of living, and showed no special dependence on defects in the intellectual field.

4. In a comparatively small number of cases bed-wetting occurred, and there was obesity after the type of Fröhlich. In fifteen subjects disturbance of speech occurred.

5. Twenty-five children were tested bythe Binet-Simon scale and by the psychologic profile method of Rossolimos. A lowering of intellectual activity could be established which in the subcortical psychopathic cases without the parkinsonian syndrome was inconsiderable and the result of character dysfunction. In the parkinsonian form the intellectual lowering was greater, and represented a type of hypotonic dementia.

6. All neuropathologic and psychopathologic symptoms, emotional dysfunction of volition, intellectual backwardness and degeneration of the instincts which are observed in epidemic encephalitis in children in the residual stage are pathognomonic. They are bound up with an alteration of different areas of the subcortical gray matter of the brain, with the exception of the intellectual disturbance in postencephalitic Parkinson's disease, in the occurrence of which the higher cortical centers play a rôle.

EMERSON, Boston.

STATUS EPILEPTICUS TREATED WITH A DIETHYLAMIN OF DIETHYLBARBITURIC AND DIPROPENYLBARBITURIC ACIDS. C. CLEMMESEN, Ugesk. f. Læger **28**:662, 1926.

Sixty-one patients with frequent epileptic attacks were treated with a diethylamin of diethylbarbituric and dipropenylbarbituric acids with favorable results.

SCHLUTZ, Minneapolis.

LEAD CONTENT OF THE HAIR OF CHILDREN WITH SEROUS MENINGITIS AND OF THEIR MOTHERS. KATSUMI TADA, Oriental J. Dis. Inf. **1**:73, 1926.

In Japan, cases of serous meningitis due to lead poisoning are not infrequent. The use by nursing mothers of a face powder containing lead is believed to be a frequent source of the condition.

The author has analyzed the hair of the head in the patients and in their mothers for lead. The hair is cut and washed with ether; it is then reduced to ashes in an electric oven; the ash is dissolved in nitric acid and the lead is determined iodometrically after precipitation as lead chromate.

The results are shown in the following table:

	Average Per Cent	Variation Per Cent
Babies with lead poisoning.....	0.2193	0.030—0.875
Mothers of these babies.....	0.1507	0.025—0.396
Lead workers	0.0454	0.015—0.080
Normal babies	0.0056	0.001—0.017
Normal boys	0.0029	0.000—0.010
Normal girls	0.0066	0.001—0.028
Normal men	0.0052	0.000—0.022
Normal women	0.0062	0.001—0.018

The milk of the mothers of the babies with the serous meningitis was analyzed and was found to contain an average of 0.243 mg. of lead per liter (varied from translation to 0.763 mg.).

Less than 20 per cent of the lead content of the hair was found in the washings when it was soaked in ether and in 25 per cent acetic acid.

Lead is shown to be bound in the keratin containing substances of the body, and is eliminated in part by this means.

Experiments were made with a nursing goat and two kids. Lead was fed to the mother, which caused chronic lead poisoning; the hair of the kids showed lead in larger amounts than did the hair of their mother. The hair of control animals contained no lead.

THE OSMOTIC PRESSURE OF THE CEREBROSPINAL FLUID. INFLUENCE OF THE OSMOTIC PRESSURE OF THE FLUID ON THE BODY TEMPERATURE. J. KENEKO, Oriental J. Dis. Inf. **1**:79, 1926.

The author studied the effect of variation of the osmotic pressure of the cerebrospinal fluid on the temperature of rabbits. Solutions of glucose of varying strengths were injected intraspinally to cause changes in body temperature. Hypertonic solutions caused general disturbance in the animal as well as increased temperature. Hypotonic solutions caused no symptoms except an increased temperature. The author concludes that intraspinal medication should always be isotonic.

HIGGINS, Cincinnati.

Diseases of the Eye

OPHTHALMOMYIASIS SUBRETINALIS. W. P. C. ZEEMAN, Nederl. Tydschr. v. Geneesk. **70**:1192, 1926.

The name ophthalmomyiasis externa or interna is given to the condition in which larvae of flies are found in the conjunctival space or in the eye. The author describes a case, in which a *Dipiera* larva was found in the subretinal space in the enucleated left eye of a boy, aged 6 years. The author assumes, on the grounds of his microscopic observations, that the larvae must have perforated the sclera, and must have reached the corpus vitrium along and through the zonule of Zinn; by partly destroying this, they then reached the already loosened retina.

VAN CREVELD, Amsterdam.

Diseases of the Ear, Nose and Throat

THE RESULT OF IONIC MEDICATION IN CHRONIC MIDDLE EAR SUPPURATION. NORA LISTER, J. Laryng. & Otol. **41**:94 (Feb.) 1926.

It was decided to follow up those patients with chronic suppuration in the middle ear who were treated by zinc ionization at the Royal Infirmary, Edinburgh. Most of these patients had been treated previously by the ordinary syringing and administration of spirit drops.

The method of zinc ionization was, first, to syringe the ear with compound solution of cresol and then with zinc sulphate. The patient was placed on the side, with the ear to be treated on the upper side, and the canal was then filled with zinc solution. The zinc electrode was fixed to a vulcanite speculum and was attached to the positive pole of the battery. The negative pole was applied to the forearm over a thick pad of lint soaked in warm saline solution. A current of from 2 to 4 milliamperes was used for from ten to fifteen minutes (longer in the postoperative cases). This treatment was carried out weekly four or five times, spirit drops being used daily during that time without syringing. The treatment was not used on children under 5 years of age.

Of thirty-eight patients treated, in thirteen the ears were found to be dry; in three, the ears were stated to be dry; in five, the ears were improved and in three the improvement was only temporary. Twenty patients showed no improvement. The cases are classified as to causation, the site of the perforation, the presence of granulations and as to whether a radical mastoid operation had been performed. The best results were obtained in the patients in whom the cause was other than scarlet fever, diphtheria or measles, when the perforation was posteromarginal, and when there were no granulations present. In this series, 25 per cent were cured and 11.8 per cent were improved.

THE PROGNOSIS OF MIDDLE EAR SUPPURATION IN CHILDREN: A REVIEW OF FIVE HUNDRED CASES. DOUGLASS GUTHRIE, J. Laryng. & Otol. **41**:305 (May) 1926.

Otitis media is often responsible for much ill health, and may cause many secondary infections, such as gastro-enteritis and pneumonia, from infective material escaping through the eustachian tube. Otitis media is most common during the first two years of life, and more common in children several months old than in young infants. The author believes that the ordinary cold is responsible for more cases of otitis media than are the exanthems. It is pointed

out that in latent otitis media the otoscopic observations may not be definite, so that a diagnosis must be made of the general condition. An exploratory paracentesis is advised.

When there is acute suppuration in the mastoid and the simple mastoid operation has been performed, the wound should be packed with bismuth subnitrate, iodoform and paraffin and closed. The results are uniformly good.

The causes of chronicity are chiefly neglect of treatment and recurring colds in the head. After any operative interference, the patient should be followed for years.

There is a report made of the results of following up 500 cases from the Royal Hospital for Sick Children for the years 1920 to 1924. Of the patients with acute suppurative otitis media treated conservatively, 78 per cent were cured, whereas 75 per cent of the patients with acute mastoiditis with operation were also cured, i. e., there was no discharge or deafness. Therefore, the prognosis with mastoiditis when properly treated is as favorable, except that there were eight deaths from meningitis and sinus thrombosis in the series of patients with mastoiditis. In chronic suppurative otitis media, 57 per cent were cured by conservative treatment. In the patients in whom an operation was performed, 25 per cent of those who had the radical operation were cured, but when the simple mastoid operation was performed, 75 per cent were cured; so that in children it is obvious that the simple operation should first be attempted, unless there is marked destruction of the middle ear structures.

There were twenty-two cases of tuberculous otitis media, in all of which the diagnosis was confirmed microscopically. The radical operation was performed on every patient. In only one patient was there a complete cure.

In conclusion, the author suggests that more care should be given to protecting children from the common cold. More care should be given to nasal hygiene, especially in teaching children the proper method of blowing the nose. Infection of the teeth and tonsils should be dealt with, and patients with recurrent otitis media should be kept constantly under attention.

[ARCH. OTOLARYNG.]

SUCCESSFUL TREATMENT OF CHRONIC DISCHARGING EARS AND ALLIED CONDITIONS BY MEANS OF ZINC IONIZATION. JOHN MCCOY, Laryngoscope **36**:434, 1926.

The use of zinc ionization in the treatment of chronic suppurative otitis media has proved successful in a considerable number of cases. Its limitations, however, are well defined. McCoy states that when there exists a marginal perforation in Shrapnell's membrane with bone necrosis and foul smelling pus, this method should not be used. He has found it exceedingly useful in cases of subacute and chronic discharge in which the perforation is central or near the margin, and when numerous other methods used for chronic suppuration have failed. Extension of infection to adjacent structures should be ruled out before resorting to its use.

Ionization is a chemical decomposition effected by means of an electric current. Ions result from the electrolytic decomposition of a molecule, and are either electronegative or electropositive, the former being called anions and the latter cathions. The electropositive, or cathions, tend to flow toward the negative pole. It has been found that, by the use of a solution of sulphate of zinc at the positive pole and the ordinary saline solution at the negative pole, it is possible to drive the zinc ions into the exudate and into the membranes of the ear cavities.

The technic of the treatment consists of cleansing the ear thoroughly with warm water, after which 4 per cent cocaine solution is applied to the middle ear on a cotton pledget for from five to ten minutes. The patient is then placed on a table with the diseased ear upward. After the pledget of cotton has been removed, the ear is filled with a 1 or 2 per cent zinc sulphate solution, and the zinc electrode attached to the positive pole is placed in the ear through a vulcanite speculum so that the zinc electrode and the zinc sulphate solution are in contact. The patient is then given the other pole to hold in the hand, or it is applied to the arm, being wet with a saline solution. The current is then turned on gradually until a strength of 3 milliamperes is reached. This is allowed to continue for ten minutes, when the current is gradually turned off. Slight vertigo or a slight pain in the region of the eustachian tube may occur in some instances. The author has treated eighteen patients with this method with very gratifying results, the patient's ear condition drying up in from two to six treatments in the majority of cases. The treatments were administered once every four or five days.

A New Method of Anesthesia for Opening Peritonsillar Abscess. Gordon D. Hoople, Laryngoscope **36**:577, 1926.

There are obvious reasons why surgery for the relief of peritonsillar abscess is usually performed under local anesthesia. The results obtained from a local anesthetic in such cases, however, are, at the best, rather unsatisfactory. Realizing this fact, Hoople, after a careful study of the nerve supply of this region, decided that cocainization of the sphenopalatine ganglion might help in solving the problem. The anterior, middle, and posterior palatine nerves, all being branches from this ganglion, have some of their terminal fiber in the tonsil and in the soft palate adjacent to the tonsil. Consequently, cocainization of the ganglion on one side should diminish sensation in the soft palate of the same side, and thereby aid in securing a better anesthetization in opening a peritonsillar abscess. A short series of ten cases was tried with this method. Although there was some sensation still present at incision, the author believes the observations in each case seem to justify the procedure. The ganglion was cocainized in the usual manner, with a 10 or 20 per cent solution of cocaine. After four or five minutes, several of the patients volunteered the information that they could swallow much easier and with much less pain. Other patients agreed to this on questioning. They were also able to open the mouth much wider, thus giving the surgeon a better view in making his incision.

Snapp, Grand Rapids, Mich.

The Function of the Tonsils. Viggo Schmidt, Ugesk. f. Læger **26**:613, 1926.

Normally, the tonsils produce from the destroyed lymphocytes an antisubstance counteracting the bacterial growth in the tonsils. Massage of the tonsils will cause an increase of this antisubstance to be excreted into the blood stream, also a slight lymphopenia. The massage of chronically infected tonsils increases the polymorphonuclear cells of the blood stream.

This paper leaves the impression that changes in lymphocytes or leukocytes in the blood of the capillaries after massage is small and inconclusive in either normal or chronic tonsillitis.

Schlutz, Minneapolis.

Diseases of the Skin

A Contribution to the Study of the Virus of Herpes. (Moroccan Strain.) P. Remlinger and J. Bailly, Ann. de l'Inst. Pasteur **40**:253, 1926.

A strain of herpes virus isolated from the lip of a Moroccan was used in a large series of animal experiments. The guinea-pig was found to be less susceptible to the disease, and the symptoms are less characteristic than those produced in rabbits. By passage the virus may become very virulent for cats, and from such a passage strain it is possible to produce the disease in dogs. In rabbits, herpes of the nervous system was produced by intradermal, epidermal, subcutaneous, intraperitoneal, intralingual, intratesticular, intravenous, intraconjunctival, intranasal inoculations and by ingestion preceded by bile feeding. The virus is found in the central nervous system and in some of the large peripheral nerves, in the saliva and exceptionally in the blood. It has not been found in the pancreas, bile, bone marrow or urine. It diffuses in glycerol and impregnates normal brain tissue. It was not found possible to detect antibodies by complement fixation. There does not appear to be any antagonism between the virus of herpes and the virus of rabies; both can develop in the same brain.

G. B. Rhodes.

Neurodermatitis from Oxyuriasis: Pathogenesis of Prurigo. E. Schröpl, Dermat. Wchnschr. **83**:1083-1097 (July 24) 1926.

In the four cases of chronic prurigo described, an inherited taint (alcohol) was evident, and all revealed rebelliously persisting oxyuriasis. Schröpl assumes that the mechanical and toxic irritation from the oxyurids affected directly the nerve centers in the region, the inferior dorsal and superior lumbar portions of the spinal cord. The changes in the local pain and temperature sense corresponded to this assumption, as also the similar aspect and localization of the prurigo in all, but the most convincing proof was the response to the roentgen rays applied to the region in one case, supplementing the focal reaction after intradermal injection of oxyuris extract. The itching always became more intense under emotional stress. The roentgen rays were applied to this portion of the spine at three sittings, a strong erythema dose, a week apart. Benefit was apparent from the first, and the patient is now free from all symptoms of the disease from which he suffered for nine years. The interval since has been only three weeks, but even temporary arrest confirms the mechanism of the neurodermatitis, Schröpl insists, as he explains it.

Cushing, Chicago.

Inhibition by Procaine of Light Dermatitis and Pigmentation. S. Rothman, Strahlentherapie **22**:729, 1926.

The wheal induced in Rothman's experiments by intradermal injection of a 1 per cent solution of procaine hydrochloride modified the skin so that it did not continue to share in the dermatitis and pigmentation induced by exposure to the mercury vapor lamp rays. No other substance tested possessed this property. The skin shared in the dermatitis evenly at first, but by the second, third or fourth day the area of the wheal blanched, the skin resuming its normal aspect, and this area showed no trace of the consecutive pigmentation. He has obtained this *Aussparungs* phenomenon, as he calls it, regularly in 100 subjects tested, and the contrast was almost as decided as at first by

the sixth month in the two reexamined later. If the rays are so potent that blisters form, the procaine hydrochloride area is spared the vesicles. The procaine hydrochloride has no effect in this line if not injected until after the exposure. This confirms his assumption that it is responsible for an elective absorption of ultraviolet rays.

[Arch. Dermat. & Syph.]

Absorption of Ultraviolet Rays in Human Skin. W. Schultze and S. Rothman, Strahlentherapie **22**:736, 1926.

Continuing the research on absorption of rays by procaine, it was found that the absorption occurs under conditions which link this "sparing phenomenon" with the antirachitic action of light (Hess). It seems to involve the nonsaponifiable, acetone-soluble lipoids of the outer skin.

Cushing, Chicago.

Dermatitis Exfoliativa (Ritter) and Erythrodermia Desquamativa (Leiner). O. Ullrich, Ztschr. f. Kinderh. **40**:644, 1926.

Many cases have been described in which dermatitis exfoliativa was followed by erythrodermia desquamativa, and according to most authors the two diseases are heterogeneous. Ullrich, however, believes a priori that it is plausible that a dermatitis or pemphigus, which are the same in his estimation, may represent an irritation which in a constitutionally disposed child may lead to an excessive regeneration of the skin and thus to the picture of erythrodermia desquamativa.

In nine out of his twenty-nine cases the erythrodermia began, contrary to Leiner, with blister formation. The difference in frequency, duration, course, extent of surface covered and presence or absence of intestinal symptoms can be reconciled if one assumes a transient peculiarity of the skin of the infant and a variation of the toxicity of the etiologic agent. There might be a reduced coherence of the layers of the skin resulting in exfoliation instead of blister formation, and the toxin might affect the whole body, leading to gastro-intestinal symptoms and a failure to gain even in breast-fed babies. The constitutional factor is a status seborrheicus.

The rôle of the skin as an organ in the defense mechanism grows in significance. It is understandable that in the special physiologic condition of the infant's skin and the undeveloped capacity of the young organisms to fight against an injury locally, the skin frequently may become diseased and show many pictures which must not be too sharply outlined as variations are so common, and too strict lines of division lead to the formation of too many classifications.

Emerson, Boston.

Surgery and Orthopedics

The Treatment of Bone Sarcoma by Toxins, Radiations, Amputation or Resection. Joseph C. Bloodgood, Am. J. Roentgenol. **14**:253 (Sept.) 1925.

The author suggests intensive roentgen-ray and clinical study before biopsy, because of the frequent difficulty in diagnosis by an exploratory incision. Amputation and resection are the treatment for sarcoma. Roentgen-ray treatment and radium should be reserved for lesions in which resection cannot be performed, so as to leave a limb with good function. To date, the only patients cured by amputation have been those with the lesion below the middle third

of the femur. There is no authentic upper extremity case in which the patient was cured by amputation. There is only one authentic record of cure by toxins, and the author reports a case of periosteal tumor of the upper shaft of the humerus treated with radium, in which the patient has apparently remained well five years after treatment.

STULIK, Chicago.

HYDATID CYSTS IN CHILDREN. H. MILLS, Surg. Gynec. Obst. **42**:585, 1926.

Devé, in 1918, pointed out that the seeds of echinococcosis are sown in infancy, and it is the extreme latency of the disease which is responsible for the fact that the majority of hydatid cysts cause no symptoms until the patient has attained the age of from 20 to 40 years. In children the hydatid cyst is a simple one without complications, whereas in adults it is already an old one. The proportion of suppurating cysts rises with the age of the patient. Echinococcosis is common in South America. The author reviews a large number of cases reported from Argentina, Uruguay, Spain, Brazil, France, Australia, England, South Africa, Germany, Holland and Switzerland. In North America, only five cases of hydatid cysts in children were reported up until 1901. Since then three more cases have appeared in the literature. To these the author adds three cases, bringing the total number in children in North America to date up to only fourteen.

M. L. PARKER.

OPERATIVE TREATMENT OF PYLORIC STENOSIS. MUNSTER SCHULTE, Monatschr. f. Kinderh. **32**:243, 1926.

While Schulte believes that pyloric stenosis will usually respond to medical treatment, some patients should be operated on. For these, the Rammstedt operation is the one of choice. The operation should be performed as soon as possible; that is, while the patient is still in good condition.

GERSTLEY, Chicago.

AN ISOLATED CASE OF TRAUMATIC LESIONS OF THE PANCREAS. HANS THOMSEN, Ugesk. f. Læger **25**:602, 1926.

The diastatic activity of the urine (technic according to Wohlgemuth) may be used as an index of prognosis in traumatic injury to the pancreas. The author contends that the diastatic activity of the urine is the most sensitive test of acute pancreatic disease.

SCHLUTZ, Minneapolis.

Miscellaneous

ROENTGEN-RAY TREATMENT OF CHRONIC COUGH IN CHILDREN. MULFORD K. FISHER, Am. J. Roentgenol. **14**:244 (Sept.) 1925.

The intrathoracic conditions in children amenable to roentgen-ray treatment are thymic enlargement, mediastinitis, peribronchial adenitis and hilumitis. In this series enlarged or diseased tonsils or adenoids, pulmonary tuberculosis, unresolved pneumonia, cardiac disease, pleuritic effusion, empyema and bronchial asthma, which produce chronic cough, have been excluded or proper measures for relief have been instituted. Of twenty-two patients treated for idiopathic cough, twenty were relieved by from two to five exposures of subintensive intensity at from seven to ten day intervals. In two, the condition subsided in forty-eight hours, and no recurrence has been noted.

ROENTGEN-RAY OBSERVATIONS IN THE UNCOMMON CAUSES OF HEMOPTYSIS. D. S. BEILIN, Am. J. Roentgenol. **14**:304 (Oct.) 1925.

Phthisis and mitral disease are cited as the common causes of hemoptysis, and are not considered in the paper. The uncommon causes are lung abscess, neoplasm, foreign bodies, bronchiectasis, chronic bronchitis with asthma, pneumonoconiosis, chronic infections due to blastomycosis, streptothricosis, pneumonia, syphilis, acute lymphatic leukemia and other unspecified causes. In lobar pneumonia the amount of blood is usually sufficient only to produce a rusty sputum, but in some instances the physical symptoms are atypical, and a roentgenogram may be necessary to differentiate from phthisis. A roentgen-ray examination and a stereoscopic study of the roentgenograms will point to a pulmonary lesion and its probable nature in eliciting the uncommon causes of hemoptysis. Cultural methods must be resorted to in some of the fungi infections, because they may appear in the form of lung abscess in the roentgenogram.

ROENTGEN-RAY THERAPY IN BRONCHIAL ASTHMA. MAXIMILIAN A. RAMIREZ and LEWIS GREGORY COLE, Am. J. Roentgenol. **14**:323 (Oct.) 1925.

The possible destructive action on lymphatic channels of the lungs which must play an important part in bacterial asthma must be considered. Possibly there is an effect on the hydrogen ion concentration of the bronchial cells, or a change in the colloids of the blood as a whole. The technic used was 200,000 volts, from 6 to 8 milliamperes, at a distance of 50 cm., through 0.75 mm. of copper. Of thirty-five patients treated, the result in twenty-five was negative, five were temporarily improved, and only five showed a definite improvement continuing for a period of six months or longer. The authors feel that possibly a modification of the treatment in selected cases may be of value, judging from the definite effect noted in the few cases.

STULIK, Chicago.

THE IMPORTANCE OF CHILD WELFARE WORK TO A COMMUNITY. LINTON GERDINE, J. M. A. Georgia **15**:348, 1926.

The author discusses thoroughly the work of the Athens (Ga.) Child Health Demonstration. The importance of this work to a community is clearly brought out.

MASHBURN, Atlanta.

SECTION FOR THE STUDY OF DISEASE IN CHILDHOOD. Proc. Roy. Soc. Med. **19**, (June) 1926.

E. A. Cocrayne reported the following two cases:

Case 1.—Brittle bones and blue sclerotics in a patient, aged 11 years. The father and two brothers suffered from brittle bones also. At the age of 3½ years, the patient fell and broke her right femur. Up to this time nothing unusual had been noticed. On three subsequent occasions this bone, also the right and left legs, had been broken. The patient was admitted to the hospital because she had been growing fat. The sclerotics were blue. The lower limbs were small and short. A roentgenogram of the skull showed that the pituitary fossa was smaller than normal, and a roentgenogram of the limbs showed multiple united fractures. All the bones were slender and rarefied. The sugar tolerance was slightly increased.

Case 2.—Congenital syphilis: Cirrhosis of the liver in a boy, aged 10 years.

D. W. Winnicott reported two cases of postencephalitic hyperpnea.

H. Chodak Gregory reported a case of congenital edema of the feet in a girl, aged 5 months. Her history was negative, except that at birth both feet were noticed to be edematous. At 2 weeks of age the urine contained a moderate amount of albumin, but several specimens tested since were free from albumin. The edema never spread, but was worse in the morning. The Wassermann reaction was negative. The author thought the cause was some malformation of the veins or lymphatic vessels, or both. The prognosis was good, the patient recovering because a collateral circulation was probably established.

Donald Paterson reported four cases:

Case 1.—Fibrillation of the abdominal and shoulder girdle muscles (progressive muscular atrophy?) as a sequel to poliomyelitis.

The patient was a boy, aged 11 years. At 18 months, he had an illness said to be meningitis, after which his head dropped back, and he was unable to sit up. He gradually recovered the power to walk but lost it again after an attack of measles and pneumonia at the age of 4. He was only able to get up on the curb on his hands and knees. At this time he developed scoliosis, for which he was placed on a backboard for two years. During the last nine months, flickering of abdominal and shoulder girdle muscles had been observed; the scapulae had become winged. Possibly he had suffered from poliomyelitis. Progressive muscular atrophy is known to supervene after poliomyelitis, but the earliest age at which the author had seen it occur had been at 18 years.

Case 2.—Splenic anemia (Banti's disease) in a girl of 6 years. In addition to the splenic anemia, the child had an enlarged heart and a systolic murmur.

Case 3.—Tuberous sclerosis.

Case 4.—Familial microcephaly.

Two children in the family were microcephalic; the elder was 3½ years old; the younger was 4 months old. The circumference of the former's head was 17 inches (48.2 cm.), the latter's was 13½ inches (33.8 cm.). The author believes that it is a rare occurrence for a woman to give birth to more than one microcephalic infant at separate pregnancies.

W. M. Feldman reported a case of pyloric stenosis (pylorospasm).

WILLIAMSON, New Orleans.

ULTRAVIOLET RAYS IN PEDIATRICS. GREGORIO VIDAL JORDANA, Pediatria españ. **15**:168, 1926.

The author concludes that ultraviolet rays constitute a useful agent in the therapy of children's diseases.

The maximum activity of the wave lengths lies between 300 and 150 millimicrons. The physiologic action of the rays is characterized principally by a regulatory influence on the metabolism of calcium and phosphorus, also by stimulation of general nutrition, activation of growth, increase in erythrocytes and in hemoglobin. The rays also seem to have bactericidal action.

The author reports the cases of sixty-eight treated by ultraviolet light. These comprise cases of rickets, tetany, pulmonary tuberculosis and skin conditions. In these conditions the benefit of treatment is 100 per cent. The author has also tried the treatment for infantile asthma, but not with as certain a result as in the foregoing conditions.

SCHLUTZ, Minneapolis.

TEN YEARS IN THE NATIONAL INSTITUTE FOR MATERNAL AND INFANT WELFARE. LEOPOLD MOLL, Monatschr. f. Kinderh. **32**:387, 1926.

The institute was founded and built during the years 1912 to 1914. During the war it served as a military hospital, and only in the winter of 1915 and 1916 did it revert to its original purpose. It serves a number of functions. Not only does it provide a welfare center for infants, children and mothers, but it serves as a school for the training of infant nurses, welfare workers, midwives and physicians.

The infants' quarters can accommodate 100 infants and thirty mothers. Mothers are admitted when they still are able to nurse their babies. The majority of admissions are for nutritional disturbance, and the duration of the stay varies from thirty to sixty days, depending on whether the child was breast fed or not.

During the ten years ending Sept. 30, 1925, 4,630 children and 1,944 mothers were cared for in the institution. During the last seven years, in a total of 4,043 cases there were 717 deaths, i. e., 17.73 per cent. Of these, 260 occurred during the first few days following admission. There were two peaks in the death curve, the usual summer and the winter rise. The winter death rate was due to the respiratory infections, and the curve is more striking than the somewhat smaller and less prolonged rise in summer.

The mortality during the years 1918 to 1922 remained about the same, i. e., from 12 to 14 per cent At this time a change in the technic of the feeding lowered the rate to 8 or 9 per cent.

The institute also functions as a school, and during the year about forty young women trained in welfare work graduate. As there are always from twenty-five to thirty nursing mothers in the station, the students have the opportunity of studying the breast-fed as well as the bottle-fed baby. Courses are also given for physicians, the duration varying from two to four weeks. In all, about 700 physicians have taken these courses.

GERSTLEY, Chicago.

METHODS OF TESTING THE SENSIBILITY OF TEETH. INVESTIGATION OF THE SENSITIVITY OF HEALTHY DENTINE AND INTACT TEETH TO WARMTH. MAX SPRENG, Schweiz. Arch. f. Neurol. u. Psychiat. **18**:40, 1926.

Little work has been done on this subject. It would seem that there may be some fluctuation in sensitivity of the same tooth under varying conditions. The teeth of chlorotic girls, pregnant women and the aged appear to be more sensitive. Steiner, in 1901, found that light touch sensations were not perceived; however, he noted that these subjects were sensitive to pressure. He investigated temperature sensation by heating a thermometer in a sand bath and bringing the bulb in contact with the tooth to be tested. He found that warmth could be appreciated at 80 C. and that pain was produced at 95 C. At 5 cold was felt, but cold produced no pain even at —15. Türkheim questioned whether teeth were sensitive to temperature and said that a temperature of 27 would give rise to a sensation of pain and not heat or cold. He tested carious teeth which had been excavated but were still painless. The question arose whether carious teeth could be regarded as normal, even after a clean cavity had been prepared.

Spreng found that intact teeth and healthy dentine could endure high temperatures. He devised a testing apparatus made by attaching a small strip of copper to a cautery loop, and this, in turn to the bulb of a mercury ther-

mometer. Asbestos insulation was placed between the cautery loop and the thermometer. The writer stated that this investigation would have to be carried much further before any conclusions could be drawn; however, he was of the opinion that his instrument might be useful in the investigation of sensation for warmth in other parts of the body.

WOLTMAN, Rochester, Minn.

Society Transactions

PEDIATRIC SECTION OF THE ACADEMY OF MEDICINE OF CLEVELAND

First Regular Meeting, Feb. 10, 1926, Held at the Medical Library Auditorium

SAMUEL W. KELLEY, M.D., *in the Chair*

The following program was presented:

INAUGURAL REMARKS. By DR. S. W. KELLEY.

ERYTHEMA INFECTIOSUM (CLINICAL REPORT OF SEVENTY-FOUR CASES PERSONALLY OBSERVED). Presented by DR. T. P. HERRICK.

INTRASCALP INJECTION OF NEO-ARSPHENAMINE IN CONGENITAL SYPHILIS. Presented by DR. L. R. BRIGMAN.

Second Regular Meeting, May 7, 1926, Held at the Babies' and Children's Hospital

The program was chiefly clinical, and was presented by the members of the staff:

HEMOLYTIC STREPTOCOCCUS SEPTICEMIA OF LONG DURATION WITH RECOVERY. Presented by DR. H. J. LESLIE.

LATE RESULTS OF CALCIUM DEFICIENCY IN INFANCY. Presented by DR. J. E. MCCLELLAND.

TWO CASES OF TRICHINOSIS. Presented by DR. J. D. NOURSE.

A CASE SHOWING UNUSUAL ANOMALIES. Presented by DR. C. T. J. DODGE.

VON JAKSCH AND OTHER ANEMIAS. Presented by DR. N. C. WETZEL (by invitation).

PSEUDOPARALYSIS IN RICKETS. Presented by DR. J. D. PILCHER.

STREPTOCOCCUS VIRIDANS ENDOCARDITIS. Presented by DR. C. W. BURHANS.

THE CINEMA AS A MEANS OF RECORD AND TEACHING. Presented by DR. H. J. GERSTENBERGER.

Third Regular Meeting, Sept. 22, 1926, Held at the City Hospital

The program was presented by the division of contagious diseases of the department of pediatrics of the Western Reserve University.

STUDIES IN SCARLET FEVER. A RÉSUMÉ OF WORK DONE WITH THE DICK TEST. ACTIVE AND PASSIVE IMMUNITY AND SPECIFIC ANTITOXIN THERAPY. Presented by DRS. J. A. TOOMEY, R. M. FULLERTON, SAMUEL BRAUN, M. E. KISHMAN, I. E. HALPERIN and EDWARD DOLCH.

This article will be published in full in this journal.

THE TUBERCULIN REACTION IN MEASLES. Presented by DRS. J. A. TOOMEY and KARLA P. HAHN.

The chief reason for the finding of negative tuberculin reactions at the height of the rash in measles is inert, improperly standardized or deteriorated

tuberculin. When a standardized tuberculin was used, the patients who had positive reactions after recovery from measles had the same reaction previously at the height of the rash.

RÉSUMÉ OF CLINICAL OBSERVATIONS IN ONE HUNDRED AND SEVENTY-FIVE CASES OF INFANTILE PARALYSIS. Presented by DR. J. A. TOOMEY.

Dr. Toomey gave a review of the clinical signs and symptoms occurring in this disease. The one new point to which the speaker called attention was the fact that he had instituted treatment by baths, movements and massage in the spinal type as soon as the patient was admitted to the hospital, not waiting for the temperature to drop to normal or for the pain to disappear. There were no ill effects from this early treatment of these patients; in fact, the author thought that the patients seemed to recover from the paralysis sooner than those in whom no treatment had been instituted until all the pain and tenderness had disappeared and the temperature had become normal.

JOSEPH E. MCCLELLAND, M.D., Secretary.

CHICAGO PEDIATRIC SOCIETY

Regular Meeting, May 26, 1926

GEORGE E. BAXTER, M.D., *President, in the Chair*

THE PRESENT STATUS OF SCARLET FEVER. Presented by DR. ARCHIBALD L. HOYNE.

I think it was as far back as 1895 that Klein propounded the theory that scarlet fever was due to a streptococcus. Others who disagreed made the contention that the streptococcus was responsible for complications but not for the etiologic factor in the disease. The same argument has been going on for years ever since. Gabritschewsky produced a vaccine for active immunization about 1904, and reported much success with its use. Then there was the Moser serum which received attention in 1903. Dochez' serum is claimed to be much different, yet from what has been written about it, it seems to be somewhat similar; perhaps the chief difference is that the latter can be concentrated and standardized and is more refined. At last the etiology of scarlet fever seemed settled beyond question, yet now word comes to us from Italy that di Christina insists that scarlet fever is not due to the streptococcus.

Regarding the Dick test, an increasing number of physicians have carried out various investigations, and several reports show that from 26 per cent up to as high as 40 per cent of persons with a definite history of scarlet fever may give a positive reaction to the Dick test. A person who has given a negative reaction to the Dick test may at a later date develop a positive reaction. If some of this toxic filtrate which is used for the purpose of making the test is injected, and an erythema is produced, that erythema cannot always be blanched by means of either convalescent scarlet fever serum or by any of the scarlet fever antitoxins or by the serum from a normal subject who gives a definite history of having had scarlet fever. In some cases of scarlet fever the Dick test may be positive as early as it should be, that is, within the first five days from the onset, but it never becomes negative. All these conflicting reports make one feel hesitant about regarding a negative Dick test as indicating that the subject will never have scarlet fever.

With regard to the Schultz-Charlton test, if the convalescent serum or any of the standard antitoxin serums is injected intracutaneously, within from six to twelve hours, there should be a definite blanching of the rash about the point of injection. If the rash is not scarlet fever, no blanching should occur. If there are no hemolytic streptococci present in the throat of the patient and if the blanching test is also negative, I think it is difficult to draw the conclusion that the patient has scarlet fever, no matter how much the symptoms may resemble it in other respects.

Five doses are necessary for active immunization. The toxin dosage now recommended by the Dicks is first, 500 skin test doses; second, 1,500; third, 5,000; fourth, 15,000 and fifth, 20,000 at weekly intervals.

With regard to the protective period resulting from active immunization, I think the Dicks' conclusion is that this period will probably endure from about two years on, and that the immunity may be established within about six weeks. One difficulty is that if the patient has had a positive Dick reaction, then within six weeks afterward the Dick test should be negative if the patient has been immunized. Yet some people have reported that after as many, I think, as ten doses have been given, the patient still has shown a positive Dick reaction.

In the case of passive immunity, apparently the protection which is afforded by the injection of scarlet fever antitoxin is rather parallel with the situation to be found after the injection of diphtheria antitoxin in which the immunity may last only from two to three weeks.

Among the claims made for the use of the Dick serum is that complications are avoided, provided the serum is given early. It is apparent, from the number of cases I have seen, that such an assumption is not always justified. Another claim made is that a sharp drop in temperature follows the administration of serum. I have seen such reactions only a few times. Another point made in favor of serum therapy is that the rash fades out more rapidly. I do not know what difference that makes unless it is an indication, of course, that the constitutional symptoms due to toxemia should diminish more rapidly because the toxins are being neutralized.

Finally, the point as it occurs to me is that if the serum should act in such a way that the patient is freed from the hemolytic streptococcus early, within two weeks, then every patient with scarlet fever should receive it, whether a private patient or one in a public hospital, for regardless of what the expense of the serum might be, it would still be a big saving to the patient or to the institution if the quarantine period could be cut in half.

DISCUSSION

DR. CLIFFORD G. GRULEE: I am hardly qualified to discuss this subject because I have had practically no experience. I rarely see a patient with scarlet fever, and can discuss the subject only from a theoretical standpoint.

Regarding the question of the treatment with the serum, I do not think effective results are claimed from its use in patients who have passed a certain period; that is, two or three days after the rash has appeared little is expected from the use of the serum. It therefore seems logical that if the number of complications are reduced by the use of the serum, the death rate should decline, because most of the deaths from scarlet fever have been due to complications.

A good many points have been raised. I should like to find out whether anybody has had much experience with the serum that Larson of Minneapolis is producing. I should also like to ask whether any one has noted many complications from anaphylactic reactions from this serum. I can remember

two children I saw early in the spring about a year ago who had anaphylactic reactions; the temperature was over 106 F. and the children were in a serious condition. They both recovered, however. I do not know whether the anaphylactic reaction from this serum is any more marked than it is from diphtheria antitoxin, but it seems to me that is a point to be raised.

Another point that I think might be raised is this: I understand that the Dochez serum is not being produced to the extent that it was. I have heard recently that the Dochez serum is being abandoned rapidly, and I should like to know whether that is true or not. I understand that there is only one firm selling the Dochez serum now, and that they are thinking of ceasing its production; also that the Dick serum is practically the only one that is on the market at the present time.

DR. HENRY W. CHENEY: I think Dr. Hoyne's suggestion that the quarantine of scarlet fever patients be terminated by cultures of the throat and nose is good. It seems to me that investigations should be carried out along these lines still further, and if necessary, the health departments of the city and state should be impressed with the views on a shorter quarantine; that is, the views of the physicians who are watching many of these cases, because scarlet fever is one of the diseases that entails more hardship on the family in the way of quarantine than any disease of which I know.

I have not used the Dick or any of the other serums as a therapeutic measure in scarlet fever.

I had an interesting experience this spring in a family of five children, ranging in ages from 7 months to 7 years. The third child had a well marked case of mild scarlet fever and was not sick enough for me to give therapeutic doses, but I gave a prophylactic injection of the Dick serum to the other four; none of them developed scarlet fever, although they were closely associated with the first case. The two older children developed a marked anaphylactic reaction. They had had toxin-antitoxin treatment for diphtheria a year or two ago, which probably sensitized them so that they developed more of a reaction from the Dick serum.

I have been using the Dick active immunization serum with a number of children. Up to some months ago, I sent the children directly to the Dicks, but since the serum and the tests are now available I have been giving the serum injections myself.

With regard to the objection that Dr. Hoyne raises of the family coming back for five injections, I think that if any of my patients would come back for three injections they would come back for five.

DR. S. C. HENN: My only experience has been with the use of the prophylactic antitoxin.

I am in an institution into which from one to three patients with scarlet fever were admitted every day for two weeks. Before that time rather scattered cases had been seen. There were thirty-five children, ranging from 2 to 6 years of age, with no history of previous scarlet fever, who had been exposed. All had had diphtheria toxin-antitoxin injections from a month to a year before. These thirty-five children were given the prophylactic antitoxin, Dochez method. One child developed mild scarlet fever in about ten hours. Six children in from six to twelve hours afterward developed an elevation of temperature varying from about 100 to 102 F., and lasting for less than a day. Six days later there were six other children who developed a slight elevation of temperature and a blotchy rash that lasted for only a few hours.

I saw a family of four children, all under 6 years of age, in which one child had definite scarlet fever. I gave the antitoxin to the three contacts. The parents would not permit the sick child to be taken to a hospital. Two weeks to the day after the first child became sick, a second child developed typical scarlet fever, and three weeks from the time the first child developed it, the other two children developed scarlet fever.

DR. JOSEPH K. CALVIN: I agree with Dr. Korper regarding the mortality in scarlet fever. Most of the deaths from scarlet fever occur early in the disease (first five days), and appear to result from an overwhelming toxemia or septicemia. The mortality is especially high in the so-called toxic patients, especially in those with membranous anginas, severe nasopharyngitis and large cervical glands. These are the patients who respond most strikingly to serum treatment, either to convalescent serum or to scarlet fever antitoxin. There is a marked improvement in the patient's condition in twenty-four hours, similar to that seen in severe toxic diphtheria.

Some years ago, when I was resident physician at the Cook County Hospital for Contagious Diseases, the serums mentioned in the foregoing had not yet been used. Patients with toxic scarlet fever with the severe membranous nasopharyngitis were given diphtheria antitoxin because there was no other treatment available. Many of these patients responded so remarkably that we began to think that all with severe scarlet fever had diphtheria complicating the fever, especially those with discharging noses and membranous throats. However, many of these patients showed repeatedly negative cultures for diphtheria. This marked improvement may or may not have been due to a foreign protein reaction. In fact, many patients with streptococcic sore throats with large cervical glands and much toxicity without any rash respond in the same way to diphtheria antitoxin.

As far as I can determine, patients with mild attacks of scarlet fever who have received scarlet fever antitoxin seem to run the same course as without it. In fact, in some of the mild attacks, the fever appears to last longer when the patient has received the antitoxin. I have suspected that the antitoxin was the cause of the prolonged fever, which lasted ten days instead of three or four in the patients with mild attacks. However, unfortunately, those with mild cases may develop almost as severe complications as those with severe cases, and for that reason I am still in doubt whether to use the antitoxin in all patients or not. If I were sure that it really diminished the complications, I think it would be justifiable to use it in patients with mild attacks as well as in those with toxic cases. I think that scarlet fever antitoxin should be used to combat the toxic symptoms according to the best evidence at hand.

DR. PHILIP ROSENBLUM: I want to cite one case to bring out the point as to the time of the immunity with the convalescent scarlet fever serum.

A little boy was given the full therapeutic dose of serum at the same time that a little girl was immunized with a therapeutic dose. Both received the serum on the first day, the girl in the first twenty-four hours of the scarlet fever. She developed otitis media and mastoiditis. The boy slept in the same room as the girl, but did not develop scarlet fever until five weeks after the girl had the disease. The girl was up and about when the boy came down with a typical case of scarlet fever, and developed about as severe a case of cervical adenitis as I have ever seen result from scarlet fever. Squibb's antitoxin was used in this case.

DR. M. G. PETERMAN, Milwaukee: There is not much doubt about the question of the isolation of the hemolytic streptococcus in scarlet fever, since it

can be isolated from every case of scarlet fever if the proper technic is used. One should remember, however, that there are any number of strains of hemolytic streptococcus. Some of them may have soluble toxins and some of them may not have toxins, and certain strains may produce merely a skin rash without the regular scarlet fever, and some may produce scarlet fever without the typical rash. Patients may be immunized against any further skin rashes, and they may be immunized against the toxins.

Regarding Larson's work in Minneapolis, his principal procedure is the neutralization of the soluble toxins with olein. It simply means that the toxin is neutralized with this fat or castor oil preparation, and that it is not harmful to inject.

I found that the reaction of a mother to the Dick test had absolutely no relation to the reaction of the new-born infant. I did not find a positive reaction to the Dick test in an infant under 9 months of age, which would corroborate in a way the clinical evidence of scarlet fever and that few cases of scarlet fever are seen in children under 9 months of age. A practice is always made of allowing nursing mothers to keep their infants with them in the scarlet fever ward.

DR. JESSE R. GERSTLEY: I should like to ask whether any members know of serious results that have followed the use of the toxin for immunizing purposes. I had one child who, after the third inoculation, developed symptoms of acute articular rheumatism lasting for two days. Following the fourth inoculation, he had a much worse attack. I called up Dr. Dick to inquire as to the advisability of the fifth inoculation. Dr. Gladys Dick thought it safe to continue, but the parents did not. I must confess I was somewhat relieved myself.

Just today I gave a child his first inoculation. He dropped on the floor in a dead faint one second after the needle was introduced. Have any of the physicians here had similar experiences? Is there any record in the literature of serious results from these immunizations?

DR. FRANK W. ALLIN: I should like to ask Dr. Hoyne one question. He spoke of one half of his thousand patients leaving the hospital with the streptococci present. Has he any evidence or data that they spread the disease afterward?

DR. HOYNE: Dr. Grulee spoke about the complications. He felt that if the serum eliminated the complications, then naturally the death rate should decline because the deaths usually were due to complications. That does not exactly follow. It sounds as though it should, but I was talking of the real septic condition in which the patients die within forty-eight hours. Theoretically, of course, if the complications are eliminated, the mortality should be lowered. I have seen altogether probably more than 350 patients who have had serum inoculations, and I do not think that the serum made any difference in the mortality.

At present a concentrated serum can be obtained. That means that the serum is much more highly refined than the original serum, which was unconcentrated. The percentage of serum rashes has not exceeded 15 per cent. I believe this is due to the fact that the serum is more refined than it was at the beginning.

You may find hemolytic streptococci in a normal person, but if you have a patient who has some kind of a rash and who has no hemolytic streptococci and yet the hemolytic streptococcus is the cause of the disease, then it does not seem reasonable to believe that the condition can be scarlet fever. In every patient in the contagious hospital in whom it was thought that the condi-

tion was scarlet fever, the hemolytic streptococcus was always found in the throat when it was looked for.

In order to determine whether a sufficient amount of serum has been used, a Dick test may be made. With proper dosage the test should be negative twelve hours after the administration of serum. If the Dick is still positive, that should suggest that the patient has not received enough serum.

A few years ago, in one of the contagious hospitals during a period of several months, 33.3 per cent of all the patients with scarlet fever admitted, at the time of admission had either diphtheria or a positive culture for diphtheria. That was at a time when diphtheria was prevalent. Now diphtheria has declined to such an extent that one sees few cases of scarlet fever and diphtheria combined. Even to see patients with scarlet fever with positive cultures is unusual. But when diphtheria was extremely prevalent, this was frequent. That was the time when I thought a number of patients with scarlet fever were dying from diphtheria, because a complete diagnosis was not made.

A patient left the hospital 113 days after the onset of the disease, and five days after she had reached home her sister was admitted to the hospital with scarlet fever and subsequently died. I do not think there is any doubt that the discharge from the ear was the cause of the second case being contracted.

Convalescent serum, in my experience, appears to produce more abrupt drops in temperature than the antitoxin. However, it makes a difference as to the convalescent serum that is used. It is usually recommended that the serum, of course, be given within the first three days; that is, in three days from the time that the rash appears, and not later than the fifth day from the onset.

Book Reviews

SCIENTIFIC NUTRITION IN INFANCY AND EARLY CHILDHOOD. By STAFFORD MCLEAN, M.D., and HELEN L. FALES, B.S. Pp. 404. Philadelphia: Lea and Febiger, 1925.

This book has been written by the chief of the outpatient department of the Babies' Hospital, New York, in collaboration with a former research chemist, and contains a general discussion of the principles underlying the nutrition of infants and outlines the practical application of those principles. Unfortunately, the principles laid down as fundamental are not always followed in the practical part of the book. The discussion of the function of the individual food elements in nutrition is especially good, and is largely based on research work carried out in the Babies' Hospital.

A good deal is said about the calcium need in the diet and the especial value of milk because of its high calcium content. The interesting but untenable hypothesis is advanced that Japanese and Italians are small because they do not drink enough milk and hence get too little lime.

The subject of digestion in infancy is only fairly well covered, and certain statements are made which may be questioned. It is stated that protein is more difficult to digest than other food elements. Raw milk is said to tax the digestion less than cooked milk because of the digestive enzymes present. The presence of acid in the intestinal tract is said to increase the splitting of fats. It is stated that digestive disturbances due to overfeeding with fat are more readily remedied than those due to overfeeding with other food elements.

For the feeding of the average infant the authors advise practically the same milk formula throughout the first year. For the normal infant a mixture of equal parts of milk and water, with sufficient sugar (about 8 per cent added) to bring the caloric value to 20 calories per ounce is recommended. The formula may be varied for individual infants by changing the proportions of milk and sugar but keeping the total caloric value at 20 per ounce. Top milk mixtures which, when diluted, will contain the same fat percentage as breast milk are recommended as generally preferable to whole milk dilutions. The authors also recommend the rather extensive use of sweetened condensed milk, although in the first part of the book they point out the disadvantages of this type of food and say that it should not be used for longer than from six to eight weeks. They then go on to state that condensed milk is second only to breast milk in digestibility; that the stools of infants fed on condensed milk are uniformly good. For use in normal infants they recommend a dilution of 1 to 5 so as to give 20 calories per ounce. It is advised that solid foods be added to the diet as early as 6 months of age. Vegetables are said to be "stimulating to mineral metabolism," whatever that may mean. There is a full discussion of dried milk, evaporated milk, S. M. A., butter-flour mixtures, acid milks and various proprietary preparations. "Processed," i. e., dried or evaporated milks, are stated to be considerably more digestible than fresh milk.

There is a very sound discussion of the stools in infancy and the fallacy of unrelated stool examinations is pointed out. For constipation in breast-fed babies, supplementary feedings of condensed milk are recommended. Rather complicated decoctions of malt extract, flour and sugar are also advised. It is difficult to see what advantages these would have over plain malt extract.

The subject of constipation in artificially fed infants and older children is well covered.

The authors' conception of diarrhea is very simple. To them it is "the result of increased peristalsis due to excessive acidity in the intestines." The conventional treatment with an initial dose of castor oil followed by a starvation period is recommended. Glucose solution is given by mouth during the period of starvation. In the treatment of dysentery, a diet high in protein and free from sugar is advised. There is a good discussion of the importance of maintaining the water balance and the means of bringing this about.

A chapter is devoted to chronic intestinal indigestion, or celiac disease. Allergy to milk is considered an important etiologic factor, and a milk free diet consisting mostly of cereal, broths and butter is recommended. After a period of weeks one drop of condensed milk is added to the daily diet and the amount of condensed milk is increased a drop a day. Dark brown pigmentation of the skin is given as a frequent symptom of celiac disease. This has certainly not been generally observed.

In the treatment of marasmus, the need for high calories is emphasized. It is pointed out that breast milk often fails as a satisfactory food for these infants. Malt soup formulas, butter-flour mixtures, condensed and evaporated milk and lactic acid milk are all recommended as suitable feedings. The "excellent character of the stools" in marasmus is commented on.

The discussion of rickets conforms to the best recent work on the subject. For the feeding of premature infants, condensed milk is advised as the first food, to be altered later by the addition of whole milk, being increased by one drop additions. For diarrhea in premature infants, evaporated milk with no added sugar is advised.

The surgical treatment of pyloric stenosis is considered fully, but the medical treatment is rather discredited by the authors. They question the value of atropine in pyloric stenosis and also the use of thick feeding. The thick feedings advised by them, however, hardly contain sufficient starch to be really thick. In the postoperative treatment, diluted breast milk or condensed milk is recommended as the first feeding. The infant is not returned to the breast until five to seven days after operation. Despite this unnecessarily restricted diet after operation, the results reported are good.

In a chapter on the "Nervous Child," the subject is well treated.

An appendix to the book contains numerous tables of height and weight of children of different ages, composition of food constituents, mathematical formulas and diet lists.

DYSTROPHIEN UND DURCHFALLSKRANKHEITEN IM SÄUGLINGSALTER. IHRE BEURTEILUNG UND BEHANDLUNG MIT EINFACHEN METHODEN. VON DR. LEO. LANGSTEIN. Pp. 207. Price, 14 marks. Leipzig, Georg Thienne, 1926.

Langstein has prepared this manual as a guide book for the practicing physician. Under the heading of "dystrophies" he considers the chronic nutritional disturbances of young infants. In a brief and practical way he outlines the causes of failure of infants to thrive, and describes in considerable detail the treatment of difficult feeding cases of various types. He advises no complicated formulas, but relies on breast milk, buttermilk containing about 1.5 per cent fat, and simple dilutions of whole milk with added sugar. For very atrophic infants he recommends the addition of casein to breast milk, and mixtures of breast milk and buttermilk. The importance of treatment of the underlying causes of atrophy is emphasized. He repeatedly warns against

overfeeding. The author feels that the feeding of high fat has no more effect in increasing the immunity than high carbohydrate. No mention is made of blood transfusions as a therapeutic agent in atrophy, but small subcutaneous injections of beef serum are recommended during the stage of convalescence as a form of protein "shock therapy." Although rickets is frequently mentioned as a cause of malnutrition in infancy, the condition is classified as a constitutional disease and no mention is made of the use of cod liver oil as a therapeutic agent. For persistent anorexia the addition of hydrochloric acid to the food is recommended. Langstein considers caffein the only safe stimulant for atrophic infants. He warns against the use of camphor, which he assumes cannot be detoxified by the atrophic infant.

Overfeeding and bacterial contamination of the food are especially emphasized as causes of diarrhea, although parenteral infections receive notice. The diarrheas are classified into the fermentative and putrefactive types. Dysentery is included in the putrefactive group. In the treatment of diarrheas, a fairly long period of complete starvation is advised in all cases except in extremely atrophic infants. Cathartics are recommended in the treatment of dysentery, but not for other forms of diarrhea. Breast milk and buttermilk are the only two forms of feeding recommended, extra casein being added in the case of fermentative diarrheas and extra sugar in the putrefactive diarrheas. The serious consequences of dehydration are emphasized. The author believes oral administration of fluid is preferable to parenteral administration, and advises the use of Ringer's solution by mouth.

There is a discussion of what the author terms "Heubner-Herter Infantilism," otherwise known as celiac disease. The clinical description of this condition is excellent—better than the outline of treatment.

As a whole, the book contains little that is new, but is a good summary of the subjects treated. There are no theoretic discussions and no references. Good case reports and charts add to the value of the book from the practitioner's standpoint. There are numerous excellent photographs illustrating the characteristic appearance of infants suffering from the various types of nutritional disorders. Taken altogether, the book is rather elementary and is written for the general physician rather than for the pediatrician.

CARE AND FEEDING OF CHILDREN. L. E. HOLT, JR. ed. 13. New York: D. Appleton & Co., 1926.

This little book, now in its thirteenth edition, has been revised by Dr. L. Emmett Holt, Jr., to meet, in a practical manner, the constant advance in the care and feeding in infancy and in childhood, for the guidance of mothers throughout the land. Probably no book of this character has ever been so extensively used, and all are glad to see it continued in the new edition.

It might be safely said that next to information disseminated by the Children's Bureau of the United States Department of Labor, The American Child Health Association, and the various state and city welfare bureaus, no single publication has accomplished so much to keep the well baby well, to console the anxious mother, or to convince the mother of her limitations as this book.

Chapter three, relating to the care of the premature infant, has been revised. Most books on the care and feeding of infants, including treatment, are confusing to the parents in matters of illness.

It is to be hoped that this book will always maintain the policy of eliminating the feature of therapy.

EXPERIMENTELLE UNTERSUCHUNGEN UEBER DARMBAKTERIEN. ABHANDLUNGEN AUS D. KINDERHEILK. UND IHREN GRENZGEBIETEN. By EUGEN STRANSKY and ALFONSO TRIAS. Price, 4.20 marks. Pp. 44. Paper. Berlin: S. Karger, 1926.

This is a detailed report of the intestinal flora of seven puppies of one litter, four of which received natural nourishment and three were fed on modified cow's milk. The puppies fed on cow's milk soon showed a syndrome which closely resembled that of the "Milchnaehrschaden" of the artificially fed infants.

There was only a quantitative difference in the bacterial flora of the upper parts of the intestines. The artificially fed puppies showed more microorganisms in the smears of the intestinal content than those of the puppies fed on natural food. The older the puppies were, the more marked was the difference. Beyond the lower part of the ileum the difference in the flora was marked. In the puppies given natural food there was a preponderance of gram-positive bacilli, just as in naturally fed infants. In the intestinal content of the puppies fed on modified cow's milk, both gram-positive and gram-negative forms were present in equal number. In the artifically fed puppies there were more different forms, the sporogenous forms being much more abundant. Yeast was found in the lower parts of the intestines of the artificially fed puppies; none was found in those naturally fed.

HYGIENE SOCIALE DU PREMIER AGE. By G. LABEAUME. Price, 68 cents. Pp. 216. Paris: Masson et Cie., 1926.

This study offers a survey of the problem of preventable infant mortality, with special reference to conditions in France, comparing them with the situation in other countries. The significance of the many factors which enter into consideration is evaluated. The many measures designed to alleviate these conditions are described, together with the various social agencies working toward this end. While the author appreciates the usefulness of these activities, yet she points out that their efficacy is greatly hampered by lack of coordination of the various departments, and implies that until such efforts are centralized, satisfactory progress will not be attained.

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AMERICAN JOURNAL

OF

DISEASES OF CHILDREN

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DECEMBER, 1926

PUBLISHED MONTHLY BY AMERICAN MEDICAL ASSOCIATION, 535 NORTH DEARBORN STREET, CHICAGO, ILLINOIS. ANNUAL SUBSCRIPTION, $4.00

Entered as Second-Class Matter, January 4, 1911, at the Postoffice at Chicago, Illinois, Under Act of Congress of March 3, 1879. Acceptance for mailing at special rate of postage provided for in Section 1103, Act of October 3, 1917, authorized on June 14, 1918.

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